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STUDIES OF PUPILS OF THE PENNSYLVANIA SCHOOL FOR THE DEAF

I. AUDITORY ACUITY

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The past decade has witnessed an extraordinary development in both interest in and understanding of the deafened person's problems. Otologists now are approaching the matter with a realization that their total contribution to the subject has been relatively insignificant and that this great group of seriously handicapped persons has not received the assistance which it desperately needs. This awakened interest cannot fail to add eventually not only to the knowledge of the subject but also to the therapeutic possibilities, which are now so inadequate.

Articles appear from time to time reporting work done under the auspices of foundations or individual grants devoted to the prevention, treatment and obviation of deafness, and yet the actual help derived at the present time by persons so disabled is little more than negligible. The fact that certain types of meningitis can now be treated effectively can scarcely be claimed as a contribution by the otologist any more than the extraordinary decrease in the incidence of the harmful sequelae of scarlet fever and measles following modern methods of treatment can be ascribed primarily to interest in the patient's hearing. Prompt diagnosis and treatment of infections of the middle ear and the mastoid, though undoubtedly effective, are certainly a belated accomplishment in the history of the surgery of acute infection.

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Recognition of new causes of deafness, both in children and in adults, though offering definite promise for the future, is still a matter of real interest to relatively few individual research otologists; the diagnosis and treatment of these conditions are too time consuming and the results of therapy too disappointing to warrant the busy practitioner's sustained interest.

With prevention and alleviation of deafness in mind, many states and cities make periodic surveys of all the children of public school age with special reference to their hearing. On the basis of the findings, obvious defects, such as foci of infection, are corrected. When a beginning impairment of hearing is found the child may be accorded preferential training of some sort in addition to routine attention to diseased tonsils and sinuses. These remedial measures are undoubtedly useful, but so far little evidence has been brought forward to indicate that by these measures the total number of deafened young adults has been materially reduced. In a large proportion of the cases hereditary deafness and otosclerosis are the diagnoses made. At the moment such diagnoses are a virtual life sentence; but fortunately the reported clinical diagnoses of these conditions are far in excess of postmortem microscopic demonstrations of their presence.

Whatever the ultimate solution of this truly serious problem may be, there still exists that appalling number of persons, estimated as high as 10 per cent of the total population, disabled by impaired hearing. Deafness is not a dramatic disability. It has none of the emotional appeal of blindness and is practically never the subject of radio broadcast or of exploitation. Schools established for deafened children, unlike hospital schools for crippled children, are constrained to accept the situation as final and must depend on the extraordinary patience and skill of their teachers to cope with the problem.

A gratifying number of states maintain schools for their deafened children, and the inevitable association of defective speech with profound deafness is now appreciated even in the lay mind as something entirely independent of actual mental ability. The deafened child is usually average mentally, and indeed in the early years, before realization of his isolated position has developed, his mentality may be above normal.

These schools are for the most part filled to capacity year after year, and teaching must necessarily be retarded and the period of education prolonged over the number of years required for the education of the child with normal hearing. To be properly effective the schools must have their pupils in residence, so that the particular system of teaching may be sustained without interruption. Careful analysis of the individual child's loss of hearing is essential in determining whether auricular training should be emphasized. Vocational training is being

featured in many of the better schools, and here again the choice of special training must be based on a knowledge of the individual child's makeup and special ability.

When the loss of hearing is complete the problem is definitely limited, but this fact must not be accepted without careful study. Residual hearing, whether by air or bone conduction or both, constitutes a challenge to every physician interested in the problem. Although analyses of these factors have been made from time to time, no careful audiometric studies of both air and bone conduction have been carried out on any considerable number of profoundly deafened children. These children are an immediate consideration, and there is little reason to think that their number will soon be reduced materially. Might not an analysis help to ameliorate the present situation, as well as provide data for future pedagogic and possible therapeutic effort?

With this idea in mind the board of governors of the Pennsylvania School for the Deaf, in Philadelphia, endorsed such a study, which was conducted by the Office of Child Hygiene Investigations of the United States Public Health Service, in collaboration with the otologic research laboratory of the Abington Memorial Hospital, Abington, Pa.[1]

MATERIAL AND METHOD

The hearing of 487 children was examined for both air and bone conduction. This is 92 per cent of the children enrolled on Oct. 1, 1937, and represents all the children present in the institution at the time of the survey. The examination was made with a Sonotone model 1 audiometer and with a Pilling-Witting auditory masker. As is to be expected, a few of the children, the younger ones especially, did not give reliable responses to some of the tests. Their records have, therefore, been excluded from further consideration in this study. With these excluded, the total material which serves as a basis for this analysis is composed of 460 audiometric records for air conduction and 454 records for bone conduction.

In the preparation of the material for statistical analysis it immediately became apparent that the arithmetical mean could not be employed as an efficient statistical constant to express the "average" threshold of hearing for any given tone. All these children had marked degrees of impairment, and consequently their auditory thresholds were concentrated at or around high intensities, and for some tones the majority of the children perceived no auditory stimulus even at the maximum intensity possible to this audiometer. As a consequence of this situation the statistical constants used in this study to express the average are "first quartile" and "median." The first quartile expresses the auditory threshold of the 25 per cent of the ears which heard best; the median expresses the auditory acuity of the 50 per cent of the ears with best hearing. Thus, if for a given tone the first quartile equals 40 decibels and the median 65 decibels, the auditory threshold of the 25 per cent of the ears with the best auditory acuity was at 40 decibels or better, and the threshold of one-half the ears was at 65 decibels or better. If, as will be often seen in the tables that follow, the first quartile

1. Miss Margaret Bodycomb, acting superintendent of the Pennsylvania School for the Deaf, and the entire teaching staff cooperated while this survey was being made. Mr. James Eike made the audiometric studies.

is indicated with an asterisk (not heard), it means that less than 25 per cent of the ears were able to hear the tone or, conversely, that more than 75 per cent of the ears were unable to hear the tone at the maximum intensity possible to the audiometer. Similarly, if the value of the median for a given tone is omitted, it means that less than 50 per cent of the ears were able to perceive the tone at the greatest intensity this audiometer can produce.

HEARING BY AIR AND BONE CONDUCTION

The first quartile and median auditory thresholds of the right and the left ears of these children are shown in table 1 and chart 1. From

TABLE 1.—*First Quartile and Median Auditory Thresholds of 460 Children*

Auditory Frequency, Cycles	Air Conduction, Db.				Bone Conduction, Db.			
	First Quartile		Median		First Quartile		Median	
	Right Ear	Left Ear	Right Ear	Left Ear	Right Ear	Left Ear	Right Ear	Left Ear
128	59.6	58.0	68.7	68.2	29.9	29.7	34.4	33.5
256	71.9	71.6	82.5	81.4	35.7	35.3	41.1	40.4
512	84.1	82.1	95.3	93.6	52.1	52.5	57.4	57.5
1024	92.0	89.8	*	*	*	*	*	*
2048	*	92.1	*	*	*	*	*	*
4096	93.6	91.7	*	*	*	*	*	*
8192	*	*	*	*	*	*	*	*
Number of children	460				454			

* Not heard.

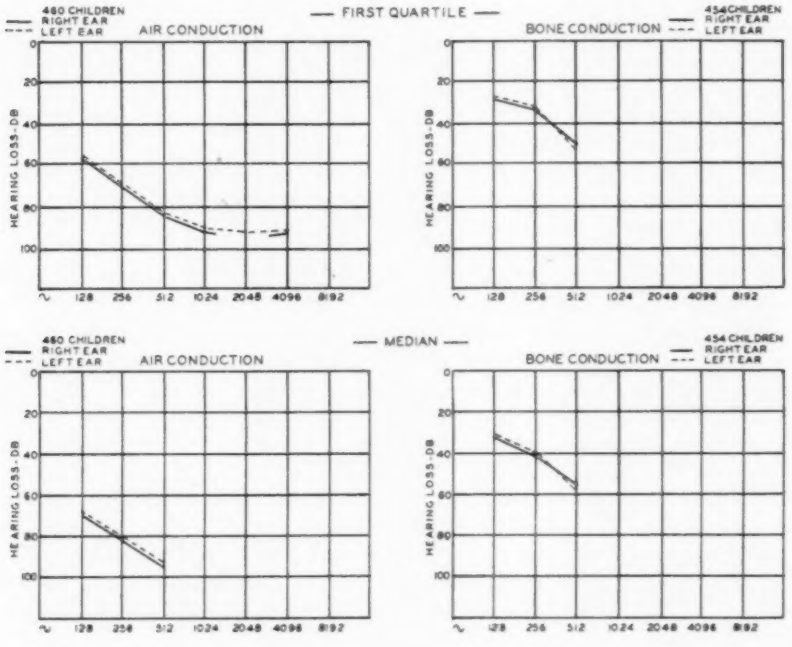

Chart 1.—Audiograms of the data presented in table 1: auditory thresholds for air conduction of 460 children and for bone conduction of 454 children.

the table it is seen that at the maximum intensity of the audiometer less than 50 per cent of the ears heard 1024 cycles or above by air conduction and less than 25 per cent heard this or higher auditory frequencies by bone conduction. Less than 25 per cent of the ears perceived 8192 cycles by air conduction at that intensity.

It is to be observed that for both the right and the left ears and for both air and bone conduction the average threshold increases as one proceeds from the low to the high auditory frequencies. This audiogram pattern, or type, is not simply an average result but is characteristic of the great majority of the individual audiograms in the sample. Such a type of audiogram is not commonly seen among the general run of cases of impairment acquired later in life. The findings in this material could, therefore, be interpreted as the consequence of either or both of the following factors: 1. The selection of the children of the school. Although admission to the school is not predicated on a previous audiometric examination, it may be that the almost complete loss of hearing for high frequencies makes the need for special instruction felt with greater urgency than would be the case if only tones of the low and middle range were involved. 2. The pathologic condition of the auditory mechanism. It may be that the deafness here discussed is accompanied in the majority of cases by the same type of defect of the inner auditory apparatus. Without other data at hand one can do no more than outline these possible factors.

It is noteworthy also that among these children all but 21 were able to perceive by air conduction, in one or both ears, at least one of the frequencies tested. There were 50 children who in one ear were unable to perceive any of the frequencies but responded to at least one in the opposite ear. In all, then, 95 per cent of the children examined could perceive at least one frequency by air conduction in one or the other ear, and 85 per cent perceived at least one in both ears. Thus, it may be said that among these children, institutionalized because of marked impairment of hearing, there were only a relatively small number that did not derive some sensation from the auditory stimulus given with the audiometer. Whether this sensation is always auditory is debatable, but these findings suggest that *total* impairment is infrequent. This, one feels, should stimulate further research in therapy and rehabilitation of these children.

From table 1 it will be noted also that the averages for the left ears are consistently, if insignificantly, lower than those for the right ears, which indicates slightly better hearing in the left ear than in the right. Since the right ear was always tested first, this difference is probably the result of habituation. Actually there is apparent a high correlation in auditory acuity between the two ears; therefore, to simplify presentation of the data in general, the averages of the left ears only will be shown in the following tabulations.

SEX AND AGE

Among the children whose hearing records for air conduction were regarded as reliable, 273 were boys and 187 were girls; among those whose bone conduction tests were included in this material, 272 were boys and 182 were girls. No significant difference between the average thresholds for either air conduction or bone conduction acuity of the two sexes is to be noted. This is illustrated in table 2 and chart 2, which show the first quartile and the median thresholds for the left ears.

TABLE 2.—*Auditory Thresholds of the Left Ears of Boys and of Girls*

Auditory Frequency, Cycles	Air Conduction, Db.				Bone Conduction, Db.			
	First Quartile		Median		First Quartile		Median	
	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
128	57.0	58.2	67.3	68.2	30.2	28.5	33.9	33.0
256	71.0	71.7	80.3	82.7	35.0	35.9	40.2	40.6
512	81.3	83.6	93.8	93.6	51.8	53.3	57.1	57.9
1024	90.5	88.2	*	*	*	*	*	*
2048	*	89.9	*	*	*	*	*	*
4096	*	88.8	*	*	*	*	*	*
8192	*	*	*	*	*	*	*	*
Number of children	273	187	273	187	272	182	272	182

* Not heard.

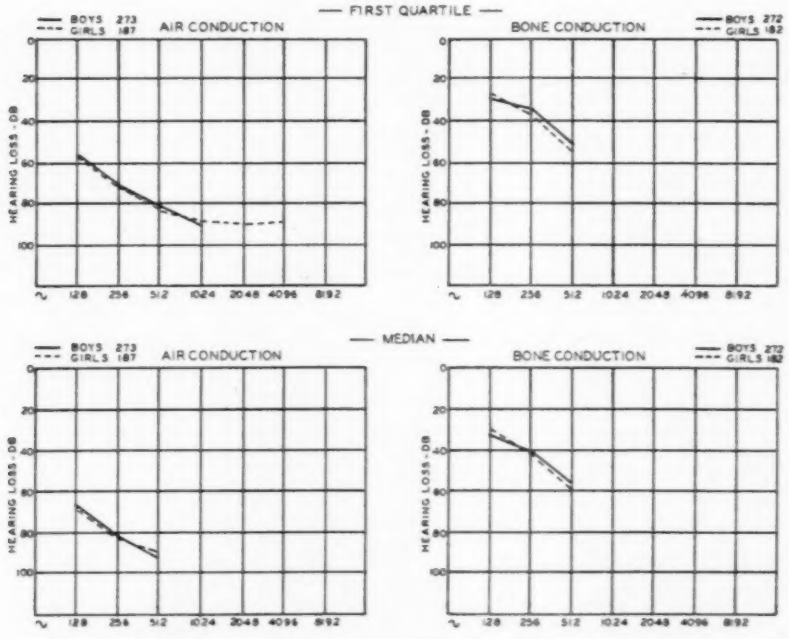

Chart 2.—Audiograms of the data presented in table 2: auditory thresholds for air and bone conduction of the left ears of boys and of girls.

The ages of the children ranged from 6 to 21 years, with a mean age of 13. When the subjects are arranged in age groups, 6 to 9 years, 10 to 14 and 15 and over, no significant differences are apparent between the average thresholds of the three groups. These tabulations have therefore been omitted. This negative finding is interesting, since it suggests that so far as this sample of children is concerned the process of impairment is not progressive.

STATED AGE AND CAUSE OF DEAFNESS

The information regarding the age and the disease associated with the onset of the impairment of hearing is derived from statements made by the parents and therefore subject to some errors. This fact must be kept in mind when evaluating these and similar observations. Of the 482 children about whom there is a report on the age of onset

TABLE 3.—*Stated Ages of Acquisition of Deafness for 482 Children*

Age, Years	Number of Children	Percentage
At or before birth	260	53.9
1	78	16.2
2	63	13.1
3	26	5.4
4	21	4.4
5	14	2.9
6	3	0.6
7	3	0.6
8	4	0.8
9	6	1.2
10	..	...
11	3	0.6
12	1	0.2
Total	482	100.0

of impairment, 260, or 54 per cent, were said to have been born deaf; 141, or 29 per cent, to have acquired the impairment before the third year of age, and the remainder, 17 per cent, at 3 years or over. The distribution of the age of onset is shown in table 3.

It will be observed that the frequency of onset decreases rapidly and fairly smoothly with increasing age. The form of the distribution curve in part may be due to the selection of the children for this school. Children who have learned to speak before impairment sets in do not need the special training that so-called mute children do. On the other hand, it does not seem that selection can account for the shape of the distribution curve for the ages up to 3 years, or even up to 6 years. The high percentage of supposedly congenitally deaf children points either to an inherited or to an acquired (in utero) inferiority of the auditory mechanism, and the rapid decrease of percentage frequency with age appears to indicate the early failure of such inferior organs.

The average auditory thresholds of the children when they are classified into three broad groups according to age at the onset of deaf-

TABLE 4.—*Stated Age of Acquisition of Deafness; First Quartile and Median Thresholds of the Left Ears of 455 Children*

Age of Acquisition of Deafness	No. of Children	128 Cycles	256 Cycles	512 Cycles	1024 Cycles	2048 Cycles	4096 Cycles	8192 Cycles
		Air Conduction—First Quartile, Db.						
At or before birth.....	247	58.2	71.9	82.7	91.3	94.1	92.4	*
Before 3 years.....	133	57.6	72.2	82.8	88.3	89.0	88.1	*
3 years and over.....	75	50.8	64.7	77.9	86.8	89.8	94.6	*
		Air Conduction—Median, Db.						
At or before birth.....	247	68.0	81.4	93.1	*	*	*	*
Before 3 years.....	133	67.4	80.9	93.6	*	*	*	*
3 years and over.....	75	67.1	81.4	*	*	*	*	*
		Bone Conduction—First Quartile, Db.						
At of before birth.....	246	30.1	34.9	52.4	*	*	*	*
Before 3 years.....	130	30.2	36.2	52.2	*	*	*	*
3 years and over.....	74	27.3	34.7	53.6	*	*	*	*
		Bone Conduction—Median, Db.						
At or before birth.....	246	33.6	39.8	56.9	*	*	*	*
Before 3 years.....	130	33.8	40.9	57.9	*	*	*	*
3 years and over.....	74	32.7	42.1	58.6	*	*	*	*

* Not heard.

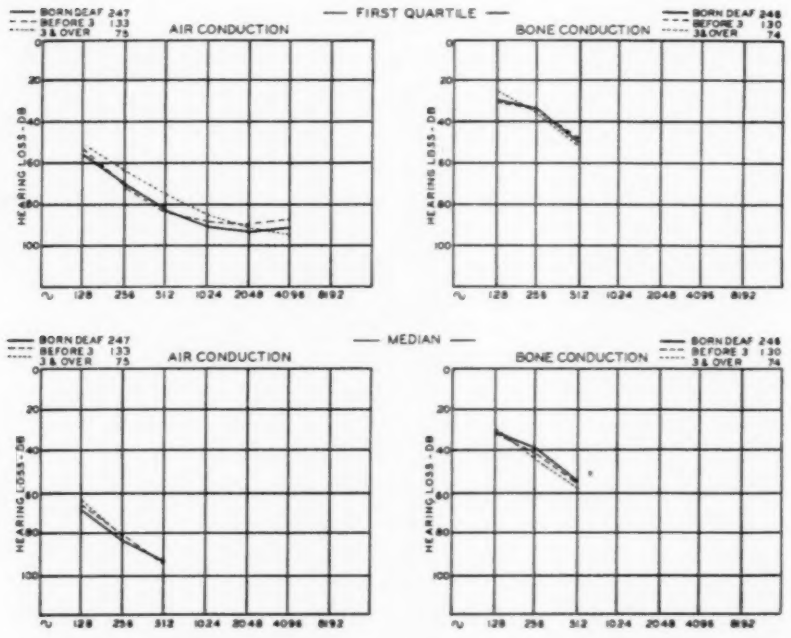

Chart 3.—Audiograms of the data presented in table 4: auditory thresholds for air and bone conduction according to stated age of acquisition of deafness.

ness are shown in table 4 and chart 3. It is seen that with respect to air conduction the first quartile threshold for frequencies up to 1024 cycles is generally lower for the children who acquired the impairment at 3 years of age and over. However, when the number of cases is taken into account, the differences between the first quartile thresholds of the three groups are not found significant statistically. The medians are practically identical in all three groups for both air and bone conduction. This suggests, if one assumes a close relation between otic lesion and auditory threshold, that the pathologic mechanism has been the same in the children who suffered marked loss of hearing after birth as in the children with a congenital defect. It therefore strengthens the suggestion resulting from the consideration of the age frequency of onset of deafness, to wit, that the defect acquired in the first few years of life may be simply a postponed manifestation of an originally inferior mechanism.

Among the 227 children who presumably acquired their difficulty in hearing after birth, the disease most frequently stated to have been the cause of the deafness was meningitis, not qualified otherwise. This disease was given as the cause of deafness in 38, or 17 per cent, of the children. Next in order come injury to the head (given as the cause for 14 per cent), measles (9 per cent) and otitis media and scarlet fever (6 per cent each). With less frequency, the stated causes include influenza, convulsions, head cold, rickets, pneumonia, diphtheria, whooping cough and poliomyelitis. Syphilis was reported for only 3 children and mumps for none. There is apparently no association between auditory thresholds and stated causes of deafness.

The wide variety of causes associated with the marked loss of hearing characteristic of those children who became deaf not long after birth, the distribution of the ages of onset and the close resemblance of the auditory threshold pattern of the children grouped according to cause and age of onset all seem to point toward the importance of the biologic constitution as a factor in the causation of this form of deafness. Bearing on this point are the results of the numerous studies on the inheritance of deafness.

From all this it seems safe to suggest that the causative factor of the type of defect manifest in this group of children should be looked for not in the experience of the child after birth but either in the uterine life or in the elements transmitted to him by his parents.

VARIATION IN ACUITY OF HEARING BY BONE CONDUCTION

The acuity of hearing by bone conduction, because of its importance as an indication of the pathologic type of the ear, deserves to be given further consideration, so that it may be possible to obtain a more clearly

defined picture of the status of the hearing of these children—one which will point toward an efficient method of studying the problems of therapy and education.

In a report of the results of studies of bone conduction on this group of children the question naturally arises as to the actual nature of the response. Was it in every instance true perception by bone conduction, or at the high intensities of the lower frequencies was it purely tactile? Experience with the particular bone conduction receiver used indicates definitely that at maximum intensity of the frequencies 128, 256 and 512 cycles there is a definite tactile factor. However, the averages shown in the following tables are all below this maximum intensity, and at the particular values for 512 cycles the tactile factor of the receiver is negligible. If, therefore, there is a response to the frequency, 512 cycles, it seems justifiable to assume that bone conduction is present.

TABLE 5.—*Number of Children Who Perceived by Bone Conduction Up to and Including the Specified Frequency*

Auditory Frequency, Cycles	Number of Children	Number Perceiving Specified Frequency in		
		Both Ears	Right Ear	Left Ear
8192	18	10	6	2
4096	9	3	4	2
2048	8	2	4	2
1024	96	44	29	23
512	264	132	54	58

The importance of adequate masking has of course been recognized, and in every instance indicated the opposite ear has been appropriately masked with the instrument mentioned in the introductory paragraph on method.

Because of the pattern common to the audiograms of these children, namely, an increase in threshold as one proceeds from the low to the high frequencies, it seems desirable to investigate the variation in the acuity of hearing by bone conduction of this sample of children from the standpoint of the highest tonal frequency perceived. In table 5 is shown the number of children who perceived, at least at the maximum intensity of the audiometer, the various frequencies tested, from 512 to 8192 cycles.

It will be seen that 397 of the 454 children perceived by bone conduction at least as high as 512 cycles in one or both ears. Especially, it is to be noted that 18, or 4.5 per cent, of these 397 children heard all the frequencies tested by the audiometer in one or both ears. If it is assumed that perception by bone conduction is essentially associated with the status of the inner auditory mechanism, it is not unlikely that these 18 children have a pathologic condition different from that of the 57

children who were unable to perceive even 512 cycles by bone conduction in either ear at the maximum intensity of the audiometer. An appraisal of the difference in air and bone conduction of the ears

TABLE 6.—*Median Thresholds for Hearing by Bone Conduction of the Ears Which Perceived by Bone Conduction Frequencies of 512 or More Cycles*

Highest Frequency (Cycles) Perceived by Bone Conduction	Number of Ears	Median Threshold at Specified Frequency, Db.						
		128	256	512	1024	2048	4096	8192
8192	28	15.0	22.5	37.5	47.5	46.7	41.0	35.8
4096	12	25.0	33.3	46.7	55.0	60.0	48.3	*
2048	10	25.0	35.0	52.5	65.0	62.2	*	*
1024	142	34.0	38.3	51.8	60.5	*	*	*
512	416	33.9	40.3	56.8	*	*	*	*

* Not heard.

TABLE 7.—*Median Thresholds for Hearing by Air Conduction of the Ears Which Perceived by Bone Conduction Frequencies of 512 or More Cycles*

Highest Frequency (Cycles) Perceived by Bone Conduction	Number of Ears	Median Threshold for Air Conduction at Specified Frequency, Db.						
		128	256	512	1024	2048	4096	8192
8192	27	61.3	67.5	64.5	63.5	72.5	58.8	64.2
4096	12	65.0	65.0	80.0	90.0	77.5	80.0	83.3
2048	9	68.8	77.5	87.5	93.8	*	*	*
1024	132	61.0	75.2	87.3	*	*	*	*
512	403	69.5	83.1	*	*	*	*	*

* Not heard.

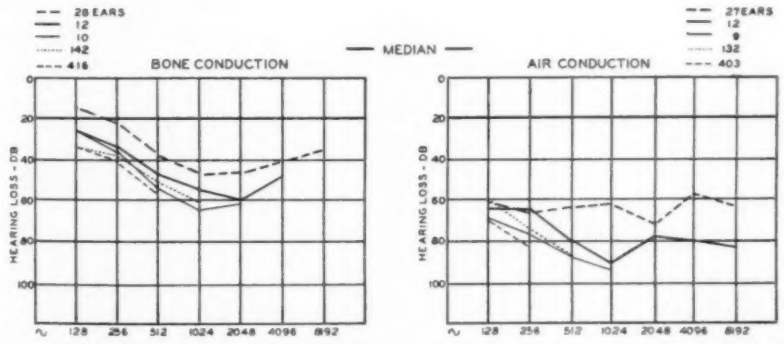

Chart 4.—Audiograms of the data presented in tables 6 and 7: relation of hearing by air and by bone conduction.

classified according to the highest tone perceived by bone conduction is obtained from tables 6 and 7 and chart 4.

To simplify computation, when the highest frequency perceived was the same in both ears the thresholds of both ears have been used, and when the highest frequency perceived was different in the two ears the

threshold of the ear with the higher limit has been included in its appropriate group, but that of the other ear has not been included in any group. For example, the median thresholds of the 28 ears which perceived all the frequencies up to and including 8192 cycles have been computed from the thresholds of both ears of 10 children, of the right ears of 6 children and of the left ears of 2 children (table 5), but the opposite ears of the last 8 children are not included in the median thresholds for any of the other groups. From table 6, which presents the data on the acuity of hearing by bone conduction of the several groups of children, it is seen that the median thresholds for all the frequencies increase in order as one proceeds from the ears which perceived the highest frequency tested to those which perceived only as high as 512 cycles. Moreover, the median degree of impairment for the ears which perceived 8192 cycles might be termed moderate, and there were some ears, 10 to be exact, in which the bone conduction threshold did not exceed 20 decibels for any frequency. In other words, 1.6 per cent of the ears had practically normal bone conduction.

When the air conduction thresholds of these ears are considered (table 7) the same regular trend is noted as one passes from the ears which perceived up to and including 8192 cycles to those which perceived up to some lower frequency. For the air conduction, however, there are no ears in which the auditory acuity enters into the range of what might be considered only slight loss.

Altogether, the information presented in tables 5, 6 and 7 is of real importance, from the standpoint of possible therapy and of the best means of rendering these children better equipped for a place in society. There are included among these institutionalized children some the condition of whose hearing is such that it should stimulate further clinical study on the means of treating a pathologic condition which, since it probably does not involve the mechanism of the inner ear, might be amenable to some form of therapy. From the standpoint of the education of these children, the differences between those with only slight loss of bone conduction and those with marked loss have real significance. The efficiency of the methods of teaching and of the acoustic instruments used in the class rooms will be dependent on the realization of these differences.

COMMENT

The data given in this preliminary report on the pupils of the Pennsylvania School for the Deaf suggest several points of real significance and indicate the necessity of further effort to correlate the many factors involved.

If, as seems to be the case, only a small percentage of these children acquire deafness after the age of 6 years, it is of great importance to have data available to indicate the actual age of onset. Though definite estimation of hearing in an infant is admittedly difficult, complete absence of hearing can be determined however young the child may be. Valuable training time is lost if the condition is not promptly recognized, particularly since the child's disability is often considered by the parents to be lack of mentality.

The single fact that a definite percentage of the children have sufficient residual hearing to warrant some sustained therapeutic procedure has great social and economic implications. If this group is typical of all student bodies of schools for the deaf—and the assumption seems a proper one—the total number of children who might be helped would be considerable. Such a possibility should stir the interest and imagination of those qualified to attempt relief.

The large group falling into what may be termed a "biologic defect" classification also is significant in view of the interest in the remote causative factors of deafness. The possibilities of preventive medicine in this particular field seem to warrant intensive investigation, however difficult the approach may be.

In future reports the present analysis of acuity of hearing will be correlated with clinical examinations, with past and family histories, and with social adjustment and scholastic achievement, as well as with the relation between the degree of loss of hearing and the aptitude for different kinds of vocational training.

SUMMARY

1. Audiograms for air conduction of 460 pupils and for bone conduction of 454 pupils of the Pennsylvania School for the Deaf are herewith analyzed from a statistical standpoint.

2. Complete lack of response to auditory stimulation is a rare condition. Ninety-five per cent of the 460 children responded to at least one tonal frequency by air conduction in one ear, and 85 per cent, in both ears.

3. In the great majority of these children the loss of hearing is bilateral and approximately equal in the two ears. Sex is not a significant factor.

4. Since age is not related to the degree of loss, the type of deafness dealt with here does not seem to be progressive.

5. Fifty-four per cent of the children were said to have been born deaf. The percentage of children who presumably became deaf after

birth is highest at 1 year of age and decreases rapidly at each successive age. No difference in acuity of hearing could be found between the children who were born deaf and those who were reported as having acquired deafness later in infancy or childhood.

6. The deafness developing after birth has been attributed to numerous pathologic conditions, among which meningitis is the most frequent. This disease was the stated cause for deafness in 17 per cent of the children. Meningitis together with trauma of the head, measles, otitis media or scarlet fever was given as the cause of deafness for almost one-half the children. There is apparently no association between auditory thresholds and stated causes of deafness.

7. A significant number of the children examined were found to have enough residual hearing either by air or by bone conduction to warrant some rational effort toward therapeutic relief.

ACUTE OSTEOMYELITIS OF THE SUPERIOR MAXILLA IN CHILDREN

REPORT OF A CASE

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KANSAS CITY, MO.

Acute osteomyelitis of the superior maxilla is an unusual disease but presents a rather typical appearance in infants. In the recorded cases the same early signs and symptoms have been present. While a gastric disturbance may be the first sign of impending trouble, the first indication of the location of the disease is the edema and swelling of the eyelid, without involvement of the conjunctiva or lacrimal sac. These areas may show a purulent inflammation later in the course of the disease. Swelling of the periorbital tissue may be great enough to cause marked exophthalmos. There are redness, swelling, pain and tenderness over the cheek of the infected side. The inflammation goes on to suppuration, and rupture usually occurs below the inner canthus, at which site a discharging fistula remains. Marked swelling and redness over one half of the hard palate are equally common. Localization also occurs in the alveolar process or buccal fold in the region of the canine fossa. Early spontaneous rupture or operative procedures establish drainage of the infected bone and result in discharging fistulas. These multiple sinuses show a characteristic discharge of purulent material containing small particles of bone or sequestrums and sometimes unerupted teeth. These fistulas are easily probed. As a rule, there is a thick purulent discharge from the nose, although this finding has not been described in some of the reports of other observers. The temperature is most often irregular; anorexia may be noted, and there is difficulty in feeding, due to the soreness of the mouth, the swelling of the sucking pad and the presence of pus in the nasopharynx and nose. The outcome of the disease may be complete healing, healing with persistent sinus formation, development of secondary foci or death.

REPORT OF CASE

Two weeks prior to her admission to the hospital, Anita C. was delivered by cesarean section. When she was 2 days old, an abscess began to develop in the

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right postauricular region. On the seventh day, an abscess in the left buccogingival fold was found and incised. On the tenth day, the swelling of the face was marked, and a fistula appeared just below the inner canthus of the left eye. On her admission to the hospital, there was marked swelling of the left side of the face, with edema and redness of the left lower eyelid. There was a profuse purulent discharge from the fistula on the face and the three smaller openings in the alveolar ridge. The discharge from the eye was seropurulent. Thick, creamy pus was drained from the ostium of the left antrum. The infant was poorly nourished and sick. The umbilicus was infected. Both the liver and the spleen were palpable below the costal margins. There was a papular eruption over the chest and the

Photographs of the patient; *A*, on admission; *B*, approximately four and a half months later; *C*, about six months later, our last contact with the child.

flexor surfaces of all extremities. The temperature was 101 F. The white cell count was 13,500. Examination of the urine showed no abnormality. A blood culture was negative.

With the patient under general anesthesia, a small antral window was made under the left inferior turbinate. A probe placed in the antrum made contact with two other probes, one of which was passed through the fistula on the face and the other through one of the sinuses of the alveolar process. We concluded that the antrum had been involved by direct extension from the tooth buds. The ophthalmologist had suggested the ethmoid cells as the primary focus of the infection, but no evidence was found that would bear out this theory. After

this drainage the patient improved rapidly and three months later was perfectly well. The sinus of the alveolar process had not completely closed, but the one on the face was entirely healed.

COMMENT

Although we concluded at the time of operation that the osteomyelitis was due to direct extension from the antrum, a more complete history obtained later pointed to the hematic origin of the infection. The surgeon who had performed the cesarean section on the mother assured us that the child had not been traumatized in any way during the operation. The physician who had charge of the baby during its first two weeks stated that the abscess behind the right ear made its appearance on the second day and was followed by similar lesions on both breasts on the fourth day. The swelling of the left side of the face, cheek and hard palate was noted on the seventh day, and drainage was established through the mouth. In spite of free drainage, the fistula under the inner canthus appeared on the tenth day. The nasal signs and symptoms were not apparent until after this occurrence. The presence of the infection elsewhere in the body previous to the onset in the upper jaw, the fact that there had been trauma and the late appearance of the nasal discharge, all point to the hematic origin of the infection.

The alveolar process is the location of choice for drainage. An incision through the skin overlying the infraorbital ridge always leaves a disfiguring scar and so is to be avoided whenever possible. While prolonged drainage through the mouth usually results in the loss of several tooth germs and necessitates the use of a dental plate later, it does give a good cosmetic result and is therefore preferable. In our case the fistulous tract through the skin at the inner canthus was already present, and scarring was unavoidable. The infraorbital ridge was destroyed by the disease, and a depressed scar involving the entire cheek resulted. Plastic repair will be necessary later.

ANATOMIC CONSIDERATIONS

There is more or less controversy over the cause of acute osteomyelitis of the superior maxilla in infants. A clear conception of the anatomic structure of the antrum of Highmore and the superior maxilla is therefore necessary. According to the textbook of Bailey and Miller, the superior maxilla is derived from the first branchial arch. The first branchial arch divides into two portions; one of these, the maxillary process, gives rise to the upper jaw and much of the upper lip and face. At birth the body of the superior maxilla is made up almost entirely of the alveolar process, the sockets of the teeth being nearly in contact with the floor of the orbital cavity. The tooth buds or sockets in many

instances are larger than the antrum. The early maxillary antrum or sinus is a slitlike cavity in the membranous lateral wall of the nose. By a simultaneous process of resorption of the surrounding bone the primitive cavity expands more and more into the body of the maxilla. The size and shape of the maxillary sinus at birth vary considerably, as is evidenced by a study of a large number of cases. The ventrodorsal measurement is always the greatest, and the cephalocaudal measurement is intermediate between the former and the mediolateral measurement. Schaeffer gave a table of measurements for the maxillary sinus in children. For the newborn the measurements are 7 to 8 mm. in length, 4 to 6 mm. in height and 3 to 4 mm. in width. The size and shape of the antrum in the newborn make it advisable to approach through the middle meatus in an operative procedure rather than through the inferior meatus, although the careful operator can easily open into the antrum by the latter route. Onodi [1] was the first to show by his anatomic studies that empyema of the antrum of Highmore in the newborn is possible. Schaeffer has subsequently and definitely proved this point. Since there no longer can be any doubt that an infant can have empyema of the antrum, it is logical to assume that direct extension from the antrum to the body of the maxillary bone is altogether possible and does, at times, actually occur.

ETIOLOGY

It is well to consider the various avenues and modes of infection not only of the antrum but also of the maxilla, since those who disregard the antrum as the primary point of the infection in cases of this class are now in the majority. This group of physicians call attention to the extremely small dimensions of the antrum in comparison with the tooth sockets and therefore favor the maxilla itself as the initial site of infection. In support of this theory several etiologic factors may be listed, chief among them being trauma at birth, dental caries, leukorrhea of the mother, infected nipples and endogenous conditions.

The maxilla is easily traumatized during delivery, especially when use of a forceps is required. It has been pointed out that small wounds may be inflicted when the mouth of the newborn infant is being cleansed if aspiration of mucus from the nose and throat is necessary. Since infections do not enter through normal mucous membrane, such accidents account for their point of entrance into the superior maxilla. Previous writers have not accounted for the site of entry of the bacteria into the bone, but the general supposition is that it must be along the alveolar border, since it is the location in which injuries most easily and frequently occur. In the series tabulated by Marx only 1 child had been

1. Onodi, A.: Accessory Sinuses of the Nose in Children, translated by C. Prausnitz, London, J. Bale, Sons & Danielsson, 1911.

delivered with a forceps, while in 4 cases the condition was said to have been caused by infected nipples, although it is quite as logical to believe that the infection of the nipples was caused by the osteomyelitic process.

It has been suggested that an infection of the genital tract of the mother may be transmitted to the child. However, this mode of infection would be more likely to result in an intranasal inflammation than in disease of the alveolar process. Only 1 case was found in which the cultures of the pus from the fistulas of the child showed the same forms as did the smear taken from the vagina of the mother.

Several writers have been of the opinion that infection starting around the tooth buds may be the cause of osteomyelitis, just as it is in adults, and this theory is well borne out in their studies. Osteomyelitis of the maxilla is seen in adults seldom as compared with infection of the other bones of the body; while it is rare in infants, it is of much more frequent occurrence than in adults. This is accounted for, in theory at least, by the fact that in infants the superior maxilla is a full-blooded, rapidly growing bone, in which invading organisms develop readily.

It is not sufficient to speak of a focus of infection around the tooth buds; the origin of that focus must be sought, and this search will lead to a consideration of the transmigration of bacteria in the blood stream or the hematogenous origin of disease. Much more attention must be paid to the theory of endogenous origin than has been in the past. In several of the cases on record there have been positive blood cultures which investigators have taken as evidence that they were dealing with osteomyelitis rather than with sinusitis. Various case histories have revealed other foci of infection previous to the beginning of the osteomyelitic process. In both of Posey's cases the latter followed a general infection, i. e., an axillary abscess, with pneumonia and empyema. Several other writers have reported inflammation of the tooth buds in the course of sepsis or erysipelas of the newborn.

RÉSUMÉ OF THE LITERATURE

The number of cases reported in the literature is not great. Approximately only seventy odd cases of osteomyelitis of the maxilla have been recorded since 1847, which would indicate that this is an unusual disease. Owing to the confusion that existed between acute osteomyelitis of the superior maxilla and empyema of the antrum of Highmore, the early reports must be carefully studied. The so-called empyema of the antrum reported by some writers was in many instances identical with what is now known to be acute osteomyelitis of the maxilla. Rees [2] is credited

2. Rees, G. A.: On Uncommon Forms of Abscess in Childhood, London M. Gaz. **4**:859, 1847.

with the first case in the literature, which he reported in 1847 as a case of empyema of the antrum. In 1898, A. Douglas [3] reported another case of empyema of the antrum in a child 3 weeks old, but in his article he mentions the paper given by D'Arcy Power [4] six months earlier, in September 1897. Roure,[5] 1898, and Lemaitre reported abscesses of the extremities as complications. Castex [6] treated osteomyelitis of the superior maxilla, during 1901, in an infant whose mother gave a history of furuncles of the thigh following delivery and an abscess of the nipples coming on after the onset of the disease in the child. Brown Kelly [7] had in 1904 collected the reports of 17 cases, to which he added 1 case, of "The So-Called Empyema of the Antrum of Highmore in Infants (Osteomyelitis of the Superior Maxilla)." Swoboda [8] had a series of 6 cases in 1904, in 4 of which the condition was thought to be due to erysipelas while in 2 he saw the sequestration of the dental germ due to the same osteomyelitic process, which in turn was of unknown origin. Three others wrote on this subject, Dependorf [9] (1910), Guisez [10] (1912) and von Reuss [11] (1914). In 1920, Zarfl [12] divided inflammatory suppurative disease of the dental germ into two classes: phlegmonous and osteomyelitic. He carefully traced the origin, course and termination, giving particular attention to the embryology and histology of the tooth bud. Finkelstein,[13] in 1920, discussed inflammation of the tooth buds in the course of sepsis in the newborn. Collet,[14] in 1922, reported a case of fatal osteomyelitis, complicated by an abscess of the

3. Douglas, A.: Empyema of the Antrum in a Child Three Weeks Old, Brit. M. J. **1**:368, 1898.

4. Power, D.: Empyema of the Antrum in a Child Aged Eight Weeks, Brit. M. J. **2**:808, 1897.

5. Roure, M.: Osteo-periostite maxillaire et arbitraire chez un Nouveau-né; genalisation; mort, Arch. internat. de laryng. **11**:137, 1898.

6. Castex, A.: Sinusite maxillaire grave chez un nouveau-né, Rev. de stomatol. **8**:318, 1901.

7. Kelly, A. B.: The So-Called Empyema of the Antrum of Highmore in Infants (Osteomyelitis of the Superior Maxilla), Edinburgh M. J. **16**:302, 1904.

8. Swoboda: Eine als selbständig zu bezeichnende schwere Munderkrankung des ersten Säuglingsalters (die gangränose Zahnkeimentzündung), Jahrb. f. Kinderh. **60**:683, 1904.

9. Dependorf, T.: Infektiöse Osteomyelitis des Oberkiefers, Oesterr.-ungar. Vrtljschr. f. Zahnh. **26**:195, 1910.

10. Guisez: Thrombo-phlebite du sinus caverneux, Rev. de stomatol. **17**:469, 1912.

11. von Reuss, A.: Die Krankheiten der Neugeborenen, Berlin, Julius Springer, 1914.

12. Zarfl, M.: Sequestrierende Zahnkeimentzündung im frühesten Säuglingsalter, Ztschr. f. Kinderh. **25**:266, 1920.

13. Finkelstein, H., in discussion on Zarfl.[12]

14. Collet, F. J.: Sinusite maxillaire de nouveau-né, Arch. internat. de laryng. **28**:1041, 1922.

brain. Marx,[15] in 1922, summarized all the cases of this unusual disease that had been published up to that date, including 3 of his own, a total of 35. Kettner [16] reported 2 cases in 1924, in which the condition was due to mechanical injury. Vaclav in the following year saw 2 instances of this unusual syndrome in infants and reported their case records. Bäumler's [17] report in the same year is of little importance here since his article deals primarily with the lower jaw. Bronner [18] cited 40 cases of osteomyelitis of the maxilla in 1925, which he had found in the literature masquerading under various titles. In his opinion, 82.5 per cent of the attacks occur during the first two months of life, and the majority of these are in the second and third weeks. Galli,[19] in 1926, treated a child for an infection of the urinary tract due to Bacillus coli; osteomyelitis of the maxilla developed later, and in this case Galli was able to demonstrate the hematogenous origin of the complication. Paunz [20] favored the antrum of Highmore as the focus of infection, in an article written in 1925. Karplus [21] and Bertemès [22] both reported typical cases in 1927. Merelli,[23] in the same year, detailed the history of a child 3½ months old whose initial symptoms were wholly gastrointestinal in nature but in whom osteomyelitis later developed without any nasal signs or symptoms being manifest at any time. Cadenat and Cola [24] also gave 1 case to the archives of medicine, but the patient was a child 5 years old. In their opinion the prognosis is not bad when there is early surgical intervention. Marchand [25] reviewed Bronner's work

15. Marx, E.: Eye Symptoms Due to Osteomyelitis of the Superior Maxilla in Infants, Brit. J. Ophth. **6**:25, 1922.

16. Kettner, A. H.: Osteomyelitis des Oberkiefers durch mechanische Insulte im Säuglings-und Kleinkindesalter, München. med. Wchnschr. **71**:207, 1924.

17. Bäumler, O.: Beitrag zur sequestrierenden Zahnkeimentzündung im Säuglingsalter, Arch. f. Kinderh. **77**:52, 1925.

18. Bronner, H.: Zur Kieferosteomyelitis des Säuglings, Beitr. z. klin. Chir. **133**:163, 1925.

19. Galli, P.: Osteo-mielite acuta dei mascellari, Clin. pediat. **8**:604, 1926.

20. Paunz, M.: Ueber die Nekrose der Oberkiefers bei Neugeborenen und im frühesten Säuglingsalter, Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:155, 1925.

21. Karplus, D.: Ein Fall von Osteomyelitis des Oberkiefers, Wien. med. Wchnschr. **77**:1739, 1927.

22. Bertemès: L'ostéomyélite du maxillaire supérieur chez le nourrisson, Ann. d. mal. de l'oreille, du larynx **46**:587, 1927.

23. Merelli, G.: Un caso di osteomielite acuta del mascellare superiore con notevole ascesso sottorbitario in un lattante, Riv. oto-neuro-oftal. **4**:435, 1927; abstracted, Zentralbl. f. Kinderh. **22**:378, 1929.

24. Cadenat, E., and Cola, A.: L'ostéomyélite de la mâchoire supérieur chez l'enfant de 2 à 10 ans, Pédiat. prat. **23**:187, 1926; abstracted, Zentralbl. f. Kinderh. **20**:119, 1927.

25. Marchand, L.: L'ostéomyélite des maxillaires chez l'enfant, Progrès méd. **43**:529, 1928.

but added no cases of his own. Terracol,[26] in 1928, contributed a paper dealing with the clinical importance of the disease. Van Gilse,[27] in a comprehensive article, traced the anatomic development of the maxillary sinus and on this basis showed how an infection of the antrum of Highmore is more likely to result in osteomyelitis of the maxilla in infants than in adults. Bloch and Lemoine [28] encountered, in 1928, a superficial abscess of the scalp and a deeper one in the temporal area complicating acute osteomyelitis of the maxilla, with ultimate recovery. Bosse [29] found 83 cases recorded in the literature in 1931, including 3 of her own. Her paper is a thorough review of the subject and contains a complete bibliography. In some of the cases included in her list the patient had orbital cellulitis but strictly speaking did not have osteomyelitis of the maxilla. H. D. White,[30] in a paper on this subject in 1935, reported that he had found only 36 cases in the literature and added 2 case reports. In his article he summarized all cases as to causation, pathologic picture, signs and symptoms, complications and treatment. Among his conclusions he advocated early operative intervention. Bloch, Larroque and Grupper,[31] in 1936, reported a case in which osteomyelitis complicated by septicemia due to infection with Staphylococcus aureus was treated successfully with staphylococcus toxoid. Tempestini,[32] in 1936, reported a case of osteomyelitis of the upper jaw in a 2 year old child with whooping cough. Tamura,[33] in 1936, added 2 more cases to the literature.

In American literature the first mention made of this subject was in 1901, when Platt reported 1 case of empyema of the antrum of Highmore in a young infant. Mayer [34] in the same year reviewed the litera-

26. Terracol, J.: L'ostéomyélite du maxillaire supérieur chez le nourrisson, Arch. internat. de laryng. **34**:532, 1928.

27. van Gilse, P. H. G.: Zur Frage der ätiologischen Bedeutung einer Kieferhöhlenentzündung bei Säuglingen (sogenannten akute Osteomyelitis des Oberkiefers), Arch. f. Ohren-, Nasen- u. Kehlkopfh. **117**:81, 1928.

28. Bloch, A., and Lemoine, J.: A propos de la fausse sinusite du nourrisson, Arch. internat. de laryng. **34**:192, 1928.

29. Bosse, D.: Ueber die Oberkieferosteomyelitis des Säuglings und ihre Bedeutung für den Augenarzt, Arch. f. Ophth. **126**:456, 1931.

30. White, H. D.: Osteomyelitis of the Maxilla in the Newly-Born, Arch. Dis. Childhood **10**:85, 1935.

31. Bloch, A.; Larroque, and Grupper, C.: Ostéomyélite du maxillaire supérieur chez le nourrisson: Traitement par l'anatoxine staphylococcique, Bull. et mém. Soc. méd. d. hôp. de Paris **52**:455, 1936.

32. Tempestini, E.: Osteomielite ematogena del mascellare superiore, complicata da follicolite e da fistole sottorbitaria, in un bimbo di due anni, Stomatol. **34**:161, 1936.

33. Tamura, I.: Osteomyelitis des Oberkiefers beim Säugling (Mitteilung von 2 ausgeheilten Fällen), Oto-rhino-laryng. **9**:712, 1936.

34. Mayer, E.: Empyema of the Antrum of Highmore in Young Infants, M. Rec. **60**:210, 1901.

ture on maxillary sinusitis in children and detailed the case history of a 2 year old child. In 1912, Posey [35] reported 2 cases of orbital cellulitis in children due to disease of the superior maxilla and gave a fairly complete summary of the literature up to that time. In an article dealing with sinusitis of children Dean listed orbital abscess as one of the complications but in reporting his 3 cases he did not mention osteomyelitis of the superior maxilla as being present, although he stressed the fact that the antrum is of sufficient size at 6 months to harbor an infective process. In 1923, Faulkner [36] contributed 2 cases, but since in 1 of these the patient was a 12 year old child, it should not be included in our group. Hurd and Byrd stated in their discussion of Faulkner's paper that they had had similar cases of osteomyelitis. Phelps [37] reported 10 cases of orbital cellulitis in 1924 but did not say that in all of these there was accompanying osteomyelitis of the maxilla. Gittins [38] reported 2 cases of orbital cellulitis in 1925, but in his cases also osteomyelitis was not present. The year 1928 yielded several reports: Fleming [39] published a report of 2 cases met with in his practice; 1 case was encountered by Shea,[40] and Bass [41] reported 2 cases along with a review of the literature. Wallace reported 1 case in which the patient was a child 4½ months old and mentioned the 3 cases recorded by Parmelee in the previous year. The next article to be found is a case report by Roehm,[42] published in 1931. Wilensky [43] discussed the pathogenesis and treatment of the disease but offered no case reports, in 1932; however, his paper added several authors to the bibliography—Moser, S. Kakals (1899), Comby (1904), Broca (1904), Movestin (1905), Cozzoloino

35. Posey, W. C.: Orbital Cellulitis from Disease of the Superior Maxilla in Children, J. A. M. A. **59**:1020 (Sept. 21) 1912.

36. Faulkner, E. R.: Osteomyelitis Associated with Sinus Disease, Laryngoscope **33**:557, 1923; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **7**:166, 1925.

37. Phelps, K. A.: Cellulitis of the Orbit in Infants and Children, with a Report of Ten Cases, Ann. Otol., Rhin. & Laryng. **33**:1391, 1924; abstracted, Zentralbl. f. d. ges. Ophth. **14**:880, 1925.

38. Gittins, T. R.: Orbital Cellulitis Complicating Paranasal Sinus Disease in Children: Report of Cases, Ann. Otol., Rhin. & Laryng. **34**:1259, 1925; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **8**:671, 1926.

39. Fleming, T. N.: Osteomyelitis of the Superior Maxilla in Infants: Report of Two Cases, Minnesota Med. **11**:252, 1928.

40. Shea, J. J.: Osteomyelitis of an Infantile Superior Maxilla, Ann. Otol., Rhin. & Laryng. **32**:509, 1928.

41. Bass, M. H.: Acute Osteomyelitis of the Superior Maxilla in Young Infants, Am. J. Dis. Child. **35**:65 (Jan.) 1928; abstracted, Zentralbl. f. Kinderh. **22**:254, 1929.

42. Roehm, H. R.: Acute Osteomyelitis of the Superior Maxilla in a Young Infant, Am. J. Dis. Child. **42**:1171 (Nov.) 1931.

43. Wilensky, A. O.: The Pathogenesis and Treatment of Acute Osteomyelitis of the Jaws in Nurslings and in Infants, Am. J. Dis. Child. **43**:431 (Feb.) 1932.

Cases Reported Since 1922 and All Those Found in American Literature

Observer	Age	Symptoms	Cause	Bacilli	Result
Platt	.....	.....	.....	.....	.....
Mayer	2 yr.	.....	.....	.....	.....
Posey	1 yr.	Orbital cellulitis; fistula, mouth and face	Axillary abscess	.....	Recovery
	2 yr.	Same; required secondary operation	Empyema of chest	Staph. aureus	Recovery
Faulkner	19 mo.	Nasal signs; typical	.....	.....	.....
	12 yr.	Not counted	.....	.....	.....
Hurd	1 mo.	Typical nasal signs	.....	.....	.....
Byrd	6 mo.	Typical nasal signs	.....	.....	.....
	2 mo.	Typical nasal signs	.....	.....	.....
Kettner	2 wk.	Typical	Mechanical injury	.....	Recovery
	2½ yr.	Typical	Mechanical injury	.....	Recovery
Vaclav	6 wk.	Typical	.....	.....	Recovery
	7 mo.	Typical	.....	.....	Recovery
Yerger	2 wk.	Nasal signs; fistula in mouth	Canker sore in mouth	.....	Recovery
Galli	4½ yr.	Typical	Hematogenous orgin	B. coli	Recovery
Karplus	.....	.....	.....	.....	.....
Merelli	3½ mo.	No nasal symptoms; typical	.....	.....	Recovery
Collet	1 mo.	Typical	Septicemia	Staphylococcus	Abscess of brain; death
Bloch and Lemoine	.....	Typical; abscess of scalp	.....	.....	Recovery
Fleming	6 mo.	Typical; two fistulas in alveolar process; bilateral fistulas on face; no alveolar fistula	.....	.....	.....
Shea	.....	.....	.....	.....	.....
Bass	3 wk.	Nasal and typical	Impetigo neonatorum	Staph. aureus	Recovery
	10 wk.	Nasal and typical	.....	.....	Recovery
Wallace	4½ mo.	Typical	.....	.....	Recovery
Parmelee	Newborn	Typical	.....	.....	.....
	Newborn	Typical	.....	.....	Death
	6 mo.	Typical	.....	.....	Unknown
Roehm	5 mo.	Fistula, face, and alveolar fistula	Unknown	Staph. aureus	.....
Bosse	3 mo.	.....	.....	.....	.....
	6 wk.	.....	.....	.....	.....
	7 wk.	.....	.....	.....	.....
Wolf	3 mo.	Typical	Injury from forceps	Staph. aureus	Abscess of brain; death
Poncher and Blayney	3 wk.	Nasal	Nasal infection	Staph. aureus	Death
White	3 wk.	Nasal and typical	Unknown	.....	Recovery
	2 mo.	Nasal and typical	.....	Staph. aureus	Death
Bloch	7 mo.	Typical	.....	Staph. aureus	Recovery
Tempestini	2 yr.	Typical	Whooping cough	.....	Recovery
Tamura	3 wk.	Typical	.....	Staph. aureus	Recovery
	2 mo.	Typical	.....	Staph. aureus	Recovery

(1906), Gilberti (1907), Scott-Ridell (1909), Landwerhammer (1909), Fliess (1912), Rocharr (1912), Bindi (1912), J. Francasis (1914), R. Petit (1915), Landette (1916) and Neumark (1897). Wolf,[44] in 1933, offered a report of 1 case in which the condition terminated fatally. Poncher and Blayney,[45] in 1934 gave a comprehensive résumé of the subject, with special attention to the histopathologic picture in their case. They concluded that nasal infection and sinusitis may play an important role in the pathogenesis at an early age and urged that this mechanism receive adequate consideration.

We have restricted our bibliographic references as much as possible to articles written since 1922, when Marx gave his summary of the literature up to that time. While his report does not include all cases recorded previous to that year, it is still fairly accurate.

CONCLUSIONS

While our case does not allow us to draw any definite conclusions, it does permit certain conjectures as to the causation and treatment of the specific cases at hand.

1. This case lends itself to the proof of the hematogenous origin of osteomyelitis of the superior maxilla.

2. Early operative intervention is indicated. Drainage should be established primarily through the mouth and secondarily through the antrum of Highmore.

3. Whenever possible drainage through the skin of the cheek overlying the superior maxilla should be avoided, even though a fistula should be present.

4. The maxillary sinusitis is a complication of the osteomyelitis rather than a cause of it.

5. The prognosis usually is good.

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44. Wolf, I. J.: Abscess of the Brain Following Acute Osteomyelitis of the Superior Maxilla, Am. J. Dis. Child. **45**:1040 (May) 1933.

45. Poncher, H. G., and Blayney, J. R.: Osteomyelitis of the Maxilla in Nurslings and in Infants, Am. J. Dis. Child. **48**:730 (Oct.) 1934.

PENETRATION OF THE INNER EAR BY MERCUROCHROME

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It has been well established that many solutions will enter the inner ear from the middle one. In 1908 Van Rossem [1] was the first to anesthetize the vestibular apparatus by applying cocaine to the middle ear. The next data on this subject were presented by Ross and Rawson [2] in 1935 and 1936. They showed that procaine hydrochloride, hypertonic sodium chloride solution, diphtheria toxin, quinine, ethyl alcohol and methyl alcohol applied in the middle ear produced marked changes in the vestibular function. In the case of diphtheria toxin, hearing was completely destroyed.

Since the fenestra rotunda is the thinnest and the softest tissue between the middle and the inner ear, it has been assumed that it would be the main route for soluble substances. Fowler [3] has repeatedly referred to chemicals "seeping through" the round window. He placed crystals of sodium chloride and calcium chloride on these tissues of dogs. He demonstrated microscopic changes in the organ of Corti in the lower coils.

An attempt was made by Hodjakoff [4] in 1937 to show the spread of a substance from the middle to the inner ear. He used trypan blue and found that the dye passed through the round window and stained some histiocytes and macrophages. Since trypan blue remained soluble during the long laboratory processes before examination, it is impossible to say that any portion of the spread took place during life. The weeks required for the decalcification of each temporal bone gave ample time for extensive diffusion.

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† Dr. Ross died on Dec. 21, 1938.

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1. Van Rossem, A.: Onderzoek. ged. in h. physiol. Lab. d. Utrecht. Hoogesch. **9**:151, 1908.

2. Ross, E. L., and Rawson, R. W.: Absorption from the Middle Ear, Arch. Otolaryng. **22**:312 (Sept.) 1935; Effects of Bacterial Toxins in the Middle Ear, ibid. **24**:51 (July) 1936.

3. Fowler, E. P., Jr., and Forbes, T. W.: Tr. Am. Otol. Soc. **26**:171, 1936.

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METHOD

There are two requirements for marking the depth of penetration of a substance into a tissue in a certain time. First, it must be possible to make the substance insoluble and keep it so till the sections of the tissue are examined under the microscope. Second, the insoluble substance must be colored. We have

Fig. 1.—Photomicrograph of a cochlea penetrated by mercurochrome from the middle ear: *1*, fenestra rotunda; *2*, stapes; *3*, stria vascularis; *4*, cochlear nerve and ganglion; *5*, aqueductus cochlearis; *6*, bone stained.

found that mercurochrome satisfies these demands on a vital stain. It is not necrotic. It is soluble in water. It is precipitated in water by hydrochloric or nitric acid and retains a brilliant color. However, acid mercurochrome is soluble in the common dehydrating agents, and therefore its use prohibits the preparation

of the tissue in paraffin or pyroxylin blocks. It was necessary to dry frozen sections in air.

Dogs were anesthetized with injections of pentobarbital sodium. The bullae of the ears were exposed, and a small slit was made in each for the purpose of admitting a small syringe needle. With the head in such a position that the

Fig. 2.—Photomicrograph of a cochlea penetrated by mercurochrome from the middle ear; the numbering is explained in the legend of figure 1.

eustachian tube pointed upward, the bulla of the right side was filled with 8 per cent solution of mercurochrome in water. The animal's head was kept in this position for a specified period. After that time the head was turned slightly to the right, and the left bulla was filled in the same manner. At the end of the second period both bullae were washed out with 2 per cent solution of hydrochloric acid. Physiologic solution of sodium chloride containing 3 per cent hydro-

chloric acid was perfused through the carotid arteries till the outflow was clear. Perfusion with 10 per cent solution of formaldehyde containing 3 per cent nitric acid was then begun and continued for several days. Decalcification in 3 per cent nitric acid was accomplished in the usual way.

The air-dried, frozen sections of the tissues were much poorer than the usual ones prepared from paraffin or pyroxylin blocks. The most delicate structures

Fig. 3.—Photomicrograph of a cochlea penetrated by mercurochrome from the middle ear; the numbering is explained in the legend of figure 1.

were often torn, broken or missing. The destruction of the soft tissues in the vestibular apparatus was so usual that no conclusions could be reached as to the penetration in this region. Drying the sections in air shriveled the cells so that the minute structures could not be studied. However, the depth of penetration of the mercurochrome could be observed.

RESULTS

Figures 1, 2, 3 and 4 are photomicrographs of cochleas that had been penetrated by mercurochrome from the middle ear. No other stain was used. The difference in the darkness of the tissues shown in the figures was due entirely to the amount of acid mercurochrome present.

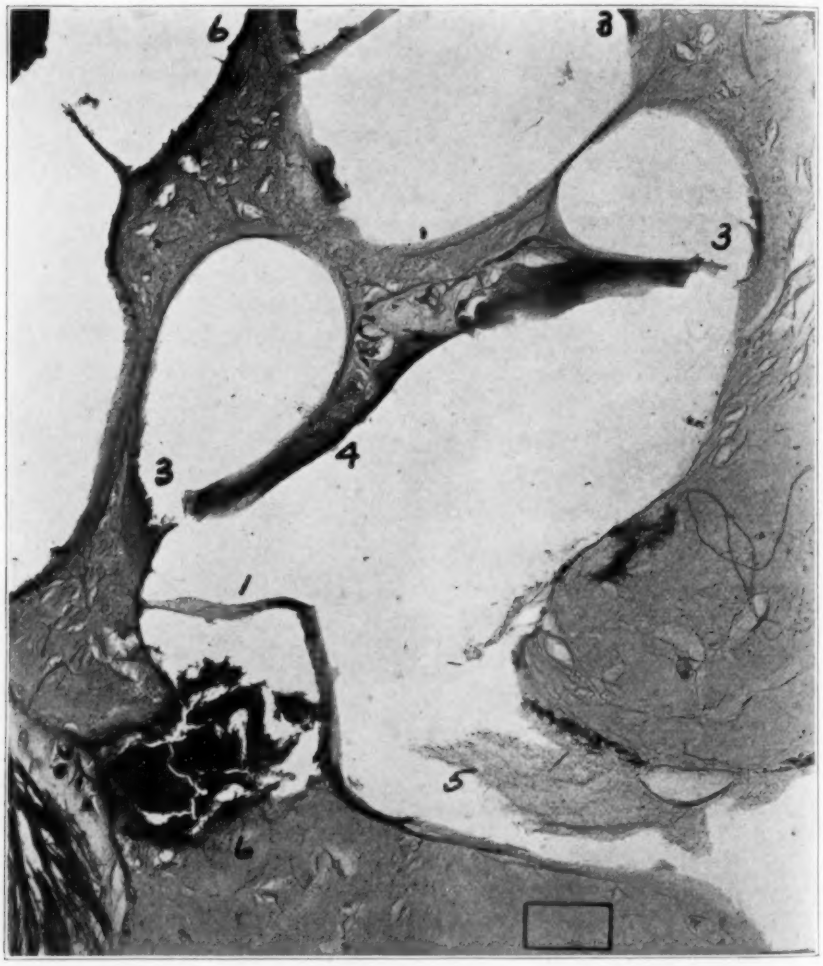

Fig. 4.—Photomicrograph of a cochlea penetrated by mercurochrome from the middle ear; the numbering is explained in the legend of figure 1.

Without mercurochrome the distinctness of the tissue would not have been more than that indicated within the small square in each figure.

The fenestrae rotundae were well stained. There were heavy deposits of mercurochrome on the side toward the middle ear but none on the other.

Observations of Staining of the Parts of the Cochlea

Animal	Period of Stain in the Middle Ear	Round Window	Articulation of the Stapes	Stria Vascularis	Spiral Lamina	Eighth Nerve and Spiral Ganglion	Aquaeductus Cochleae	Degree of Penetration of Bone
138 R	2 hr.	Very well stained inside	Stained half through	Stained	Well stained	Both well stained	Much stained over the mouth	Deep between the middle ear and the cochlea
139 R	1 hr.	Very well stained	Little stained	Well stained (more near the round window)	Destroyed	Nerve well stained	Well stained material in the mouth	Well stained over the round window
140 R	1 hr.	Very well stained	Not present	Well stained (more near the round window)	Well stained (more near the round window)	Both well stained	Not seen	Much stained around the eustachian tube
141 R	2 hr.	Very well stained	Stained entirely through to the inner ear	Well stained	Well stained	Both well stained	Faintly stained to the dura	Much over the round window
142 R	1 hr.	Stained	Not present	Stained	Well stained	Both well stained	Well stained	Much stained about the eustachian tube
143 R	1 hr.	Well stained	Slightly stained	Stained	Stained	Both well stained	Contains stain	Well stained
144 L	2 hr.	Very well stained	Mildly stained	Brightly stained	Brightly stained	Both brightly stained	Well stained	Deeply stained around the eustachian tube
144 R	1 hr.	Very well stained	Well stained	Very well stained	Well stained	Very well stained	Stained the same as the surrounding tissues	Well stained and deep penetration
145 R	2 hr.	Well stained	Well stained	Very well stained between the two windows	Well stained	Well stained	Slightly deeper stain at the mouth	Very well stained deeply into bone
145 L	1 hr.	Very well stained	Palely stained	Weakly stained	Weakly stained	Weakly stained	Mouth more stained than elsewhere	Weakly stained
150 L	20 min.	Well stained	Well stained	Stained	Stained	Stained	Stained the same as surroundings	Very well stained

The stapes was well stained, and the articulation was still more heavily stained.

The striae vasculares were well stained. The spiral ligaments were not nearly as dense.

The spiral laminas were heavily stained. This was equally true of the auditory nerve and the spiral ganglion cells.

The aquaeductus cochleae were definitely but weakly stained.

The bony wall of the middle ear was deeply stained with mercurochrome. The stain had entered this structure in large quantities and penetrated deeply.

Eleven inner ears, from 9 animals, were studied. Mercurochrome was allowed to remain in the middle ears for twenty minute, one hour and two hour periods. The observations are recorded in the table.

COMMENT

If the fenestra rotunda had been the chief avenue for the mercurochrome, the following results would have been obtained: First, the round window would have been more deeply stained than any other tissue in contact with the perilymph. This was true. On account of the thinness of the membrane it could hardly have been otherwise. Second, there would have been much of the stain immediately inside the fenestra rotunda. This condition was not seen. Third, the meshes of the aquaeductus cochleae would have been deeply stained. On the contrary, they were weakly stained. Fourth, there would have been relatively weak staining of the bony wall between the middle and the inner ear. The nerve elements, such as the fibers of the cochlear nerve and spiral ganglion cells, would have been only slightly colored. However, all these tissues were found stained with mercurochrome more deeply than any other tissue different depths of which could be observed. It was impossible to see the various degrees of staining at different depths of a membrane so thin as the fenestra rotunda.

Our observations indicate that the mercurochrome passed through all the tissues between the middle and the inner ear. The bone was permeable. The deep staining of the nerve elements could have been accomplished best through penetration of bone.

If the round window were the only or the chief route to the inner ear, an anesthetic placed in the middle ear should produce deafness first and vestibular vertigo later. In 2 human subjects we have found the reverse to be the case. A 10 per cent solution of procaine hydrochloride placed in the middle ear caused severe vestibular vertigo, but practically no loss of hearing was observed. In 1 case temporary paralysis of the facial nerve occurred.

The 11 round windows examined were well filled with mercurochrome. There was no distinct difference in the degrees of staining due to the difference in the time of the exposure to the mercurochrome. In only 1 case was there any deep stain on the inside of the fenestra rotunda.

The bones of the stapes were stained the same as the bony walls of the middle ear. The articulations were slightly more stained than the bone. However, the depth of stain was much less than that in the fenestra rotunda.

The striae vasculares were well stained in all cases. There was some evidence that those of the higher coils were less stained than the others.

The nerve elements in the spiral laminas, the spiral ganglions and the cochlear nerves were well colored. The time of exposure, from twenty minutes to two hours, seemed to have made no measurable difference.

The cochlear aqueducts were seen to be stained in all cases. The degree of staining was not sufficiently constant and deep to indicate that there was a rapid transmission of stain through the round window and out the cochlear aqueduct. The average difference between the color of the fenestra rotunda and that of the meshes of the cochlear aqueduct was great.

The bony wall of the middle ear was deeply stained to surprising depths. This is especially well shown in figure 4. The stain in large quantities had passed entirely through the wall to the coils of the cochlea. Figures 2 and 4 are sections of the same cochlea. These figures show the difference in the amount of mercurochrome present at different depths of the bone superior to the fenestra rotunda.

The changes observed by Fowler [3] in the organ of Corti after putting crystals on the fenestra rotunda are not conclusive, as he points out that the chemicals may have "seeped through" the fenestra rotunda as he has stated. However, the extraction of water from the perilymph has not been excluded as the cause for the cochlear changes.

SUMMARY AND CONCLUSIONS

Mercurochrome was placed in 11 middle ears. It was allowed to remain there twenty minutes, one hour or two hours. At the end of the specified periods the mercurochrome was precipitated by washing the middle ear and perfusing with an acid solution. Frozen sections of the acid-decalcified temporal bones were made.

Mercurochrome penetrated the entire cochlea. The tissues in the decreasing order of the depth of staining are (1) bony wall of the

middle ear, (2) fenestra rotunda, (3) nerve cells of the spiral ganglion, spiral lamina and eighth nerve, (4) stria vascularis, (5) articulation of the stapes and (6) meshes of the aqueduct of the cochlea.

The observations do not support the view that the fenestra rotunda is the chief transmitter of solutions from the middle ear to the inner ear.

Mercurochrome proved satisfactory as a vital stain.

PRIMARY MELANOMA OF THE NASAL CAVITY

REPORT OF TWO CASES

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The study of melanoma presents one of the most interesting topics in oncology. The peculiar and obscure origin of the growth and the eccentric clinical course offer a field of debate of equal interest to pathologist and clinician.

Melanoma is a malignant tumor which rarely occurs in the nasal cavity. In the English literature I could find the record of but 12 cases. Wilkinson [1] reported a case in the English *Journal of Laryngology and Otology* in 1912. He found in his research 9 other reports, of a similar nature, collected by Cozzolino [2] in 1904. In some of these early cases the absence of detailed description of the histologic observations makes it doubtful whether the condition was really melanoma. A second case was reported in the *Journal of Laryngology and Otology* in October 1930, by E. G. Collins.[3] The 2 cases which I have to present bring the number of those recorded in English literature to 14.

REPORT OF CASES

CASE 1.—A white woman aged 60 was examined in January 1931 because of recurring epistaxis from the left nasal chamber. There were no other symptoms. The source of the bleeding was found to be a small, soft, red granular wartlike growth springing from the floor of the left nasal chamber just beyond the vestibule. It was pedunculated and about 0.75 cm. in dimension. No other pathologic condition was noted in the nose or sinuses. There was no evidence of enlargement of any of the cervical glands.

The growth was removed with a snare and its point of attachment cauterized. It was studied histologically by Dr. B. L. Crawford, who submitted the following report:

"The specimen is entirely composed of masses of rather large round cells, with a small amount of stroma and a number of blood vessels. The cells forming the growth vary somewhat in size but are evidently all of the same type, and the majority contain a dark brown pigment. The growth is evidently a melanoma and, while it cannot be stated definitely whether this type of lesion is malignant or benign, has all the histologic appearance of being malignant."

Read before the College of Physicians, Philadelphia, Jan. 12, 1938.

1. Wilkinson, G.: J. Laryng. **27**:1, 1912.
2. Cozzolino, V.: Arch. f. Laryng. **15**: 77, 1904; cited by Wilkinson.[1]
3. Collins, E. G.: J. Laryng. & Otol. **45**:691 (Oct.) 1930.

The patient remained free of symptoms in the nose and throat. Sixteen months later a nodule developed in the left submaxillary region. It gradually increased in size. It was not painful or tender and was movable on palpation. It felt rather soft and cystic and was thought to be a cyst or neoplasm of the submaxillary gland. No doubt because nasal symptoms were absent and the lesion in the nose had not recurred over such a long period, the original small growth on the floor of the nose, which had been completely removed, was not thought

Fig. 1 (case 1).—Photomicrograph of a section of the primary growth (melanoma) from the left nasal chamber, showing large polyhedral cells, vascular tissue, a little stroma and many groups of cells with dark brownish pigment (× 100).

to have any connection with the tumor beneath the left lower jaw. The patient was admitted to the general surgical service of the Jefferson Hospital, and the mass was removed.

The histologic study of this tumor showed an undifferentiated type of cell with little or no stroma and with definite indication of malignancy. There was no

evidence of any pigment in the cells. The growth was thought to be a type of carcinoma when first examined by Dr. Crawford.

Postoperative roentgen treatment was given to the glands of the neck. The patient remained well for almost two years. In April 1934 deafness and tinnitus developed in the left ear. She was treated by another laryngologist, who was unaware of her previous history, for a catarrhal condition of the eustachian tubes. Two weeks later double vision and disturbance of balance developed. She was

Fig. 2 (case 1).—Photomicrograph of a section from the metastatic lesion in the submaxillary region, showing the same type of cell as is seen in figure 1 but no pigment (× 100).

again admitted to the hospital for study, and a diagnosis of intracranial metastasis of the malignant tumor from the left submaxillary region, with involvement in the region of the left cerebellopontile angle, was made. She became worse rapidly and died in a coma within six weeks.

The true course of events was not appreciated until this study was instituted. The slide from the original small melanoma in the nose was compared with the slide from the submaxillary tumor. They were found to be histologically identical

except that there was no pigment in the growth from the submaxillary region. The fatal termination was no doubt due to metastasis of the malignant melanoma of the nasal cavity, first to lymph nodes in the left submaxillary area and later to the brain.

CASE 2.—A man aged 35 was first seen in October 1934, at which time he complained of nasal obstruction on the right of four months' duration. Three

Fig. 3 (case 2).—Photomicrograph of a section from the primary growth in the right inferior turbinate, showing undifferentiated cells, with considerable degeneration and necrosis, and absence of pigment. The growth was first diagnosed as a granuloma (× 100).

years previously he had had polyps removed from the right side of his nose because of obstruction. His nose had remained clear until the onset of the present illness. In view of the nature of the lesion under discussion it is of interest to consider whether the polyps removed earlier had any relation to the subsequent tumor.

Examination revealed a granular growth involving the anterior tip of the right inferior turbinate. It was 1.25 cm. in its greatest dimension, flattened and attached by a pedicle with a rather broad base. It was firm and reddish gray, with a granular surface. It completely obstructed the anterior nares. No other pathologic condition was apparent in the nose or throat. Roentgen examination of the sinuses revealed nothing remarkable. The neck was free of palpable nodes.

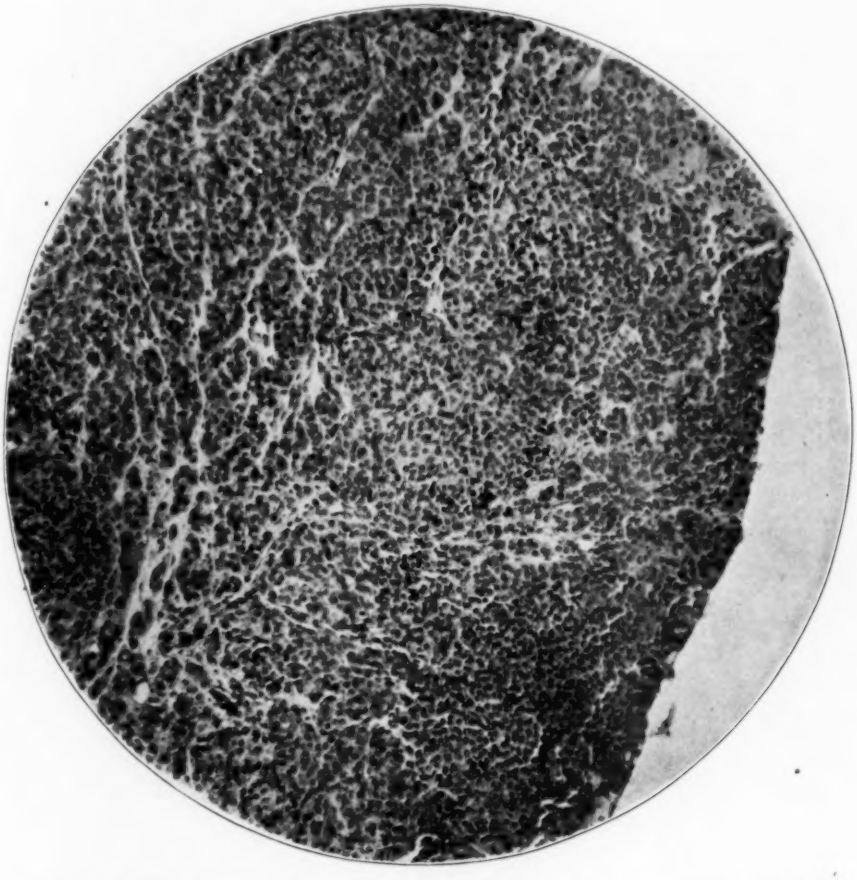

Fig. 4 (case 2).—Photomicrograph of a section from a recurrent growth in the right turbinate, showing undifferentiated cells, numerous mitotic figures, no connection with the mucous membrane and absence of pigment. The growth was recognized as a definitely malignant tumor but not as a melanoma (× 100).

The tumor was removed with a snare, with the patient under local anesthesia. The following report on the histologic picture was submitted by Dr. Crawford:

"From the cellular nature of the lesion, malignancy cannot be definitely excluded, but it is considered more likely to be a form of granuloma. It seems that carcinoma can be excluded, but the lesion may be sarcomatous."

The patient was not seen again until April 1936, sixteen months later. There was now a local recurrence of the growth, involving the anterior half of the right

inferior turbinate. The turbinate was removed for biopsy, and a report of carcinoma was given. There was no evidence of extension of the growth to the sinuses or of glandular metastasis.

A radical operation, with wide exposure and removal of the lateral wall of the nose and right ethmoid sinuses, was advised. However, operation was refused, and the patient, who lived at some distance from the city, was not seen again until ten months later, when he returned with pain in the right side of the face

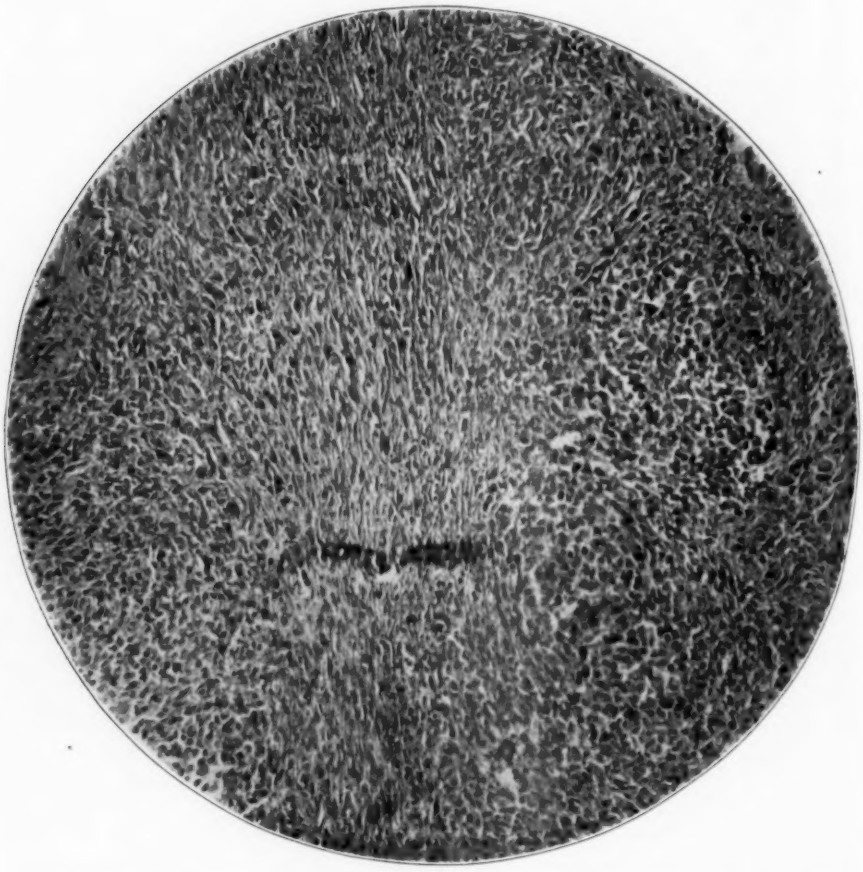

Fig. 5 (case 2).—Photomicrograph of a section from a metastatic lymph node in the right submaxillary region. The melanotic nature of the growth was recognized for the first time because pigment, though scanty, was definitely present in small groups of cells (× 100).

and a lump about the size of a walnut, firm, freely movable and not tender, at the angle of his right lower jaw.

Operation was decided on, and with the patient under anesthesia induced by avertin with amylene hydrate and ether the right external carotid artery was ligated above the superior thyroid artery and the submaxillary node removed. A Rouge, or Denker, operation was then done on the right side. This afforded

excellent exposure of the right nasoantral wall and the right antrum, ethmoids and sphenoid. The growth had extended by continuity to the nasoantral wall and ethmoid area, but the sinuses were not extensively involved.

Histologic study of the section from the submaxillary node revealed a type of cell similar to that of the primary growth on the inferior turbinate but with definite pigmentation. The diagnosis was definitely established as melanoma.

The follow-up of the patient since operation has been interesting. Some months later acute abdominal pain developed, and he was rushed to a hospital for an emergency appendectomy. An acute intestinal obstruction due to a mesenteric mass was found. The cause of the mass was thrombosis of the mesenteric vessels due to tumor cells acting as emboli.

The diagnosis of the pathologist was metastatic sarcoma. However, slides with tissue from the metastatic lesion in the abdomen were obtained and compared with the previous slides. The cells were identical and in places contained pigment.

When last heard from, two months before the time of writing, the patient was declining rapidly, with generalized metastasis. Approximately three hundred nodules had appeared over the body, with a mass in the left buttock 4 inches (10 cm.) in diameter. Roentgen therapy had no influence on the lesions.

In considering the origin of these tumors one finds a wide difference of opinion among pathologists. Evidence has been submitted in favor of four different modes of origin:

1. From the basal epithelium by a process of emigration and depigmentation (Dawson). The difficulty of uveal melanomas is disposed of in this assumption by the theory that pigmented cells of the choroid are embryologically derived from the pigmented epithelium of the retina while melanomas of the adrenal and other deep organs are assumed to rise from pigmented neuroepithelium.

2. From fibroblasts by modification (Riecke and Pini). A number of recent observers cling to the theory of endothelial origin of some melanomas while admitting that others are derived from epithelium (Lowenbach, Walsch, Herxhermer, Bowman, Moller, Fox, Johnson). They have not demonstrated specific features distinguishing the two types of tumor.

3. From the end apparatus of cutaneous sensory nerves (Masson).

4. From a highly specialized mesodermal cell, the melanoblast, or chromatophore (Ehrman, Ribbert). The chromatophore is a specific mesoblastic cell controlling production and transport of pigment, in terms of which this theory regards all manifestations of melanoma.

Ewing[4] stated that theoretic considerations favor the view that all melanomas originate from the mesoblastic chromatophore and histologic observation of human tumors that they originate from epithelial cells

4. Ewing, J.: Neoplastic Diseases: A Treatise on Tumors, ed. 3, Philadelphia, W. B. Saunders Company, 1928.

which have taken on pigmentary functions. The established tumors exhibit either carcinomatous or sarcomatous structure or both. However, the identification of this tumor with carcinoma or sarcoma is undesirable. Its cells have many specific properties which may resemble those of either epithelial or mesoblastic cells. The term melanoma should be employed to emphasize the specific character of the tumor cells. The possible origin from epidermal cells may be indicated by the term "melanocarcinoma" and the sarcomatous character by the term "melanosarcoma.

The following features were of interest in the 2 cases:

1. Ordinarily malignant melanoma is thought to have its origin in a nevus of the surface of the skin. However, it must be thought of in cases in which papilloma occurs in the anterior part of the nasal cavity, springing either from the turbinates or from the floor of the nose. The mucous membrane in this area probably has in it misplaced tissue or cell rests of cutaneous origin, which may give rise to this type of tumor. This is similar to the not infrequent origin of melanoma in the rectum, which is another area of transition from cutaneous to mucosal surface.

2. The presence of pigment in the lesion makes the histologic diagnosis easy, but its absence does not rule out melanoma. In the first case pigment was present in the primary lesion and absent in the metastatic node in the submaxillary region. In the second case there was no pigment in the primary growth on the inferior turbinate but definite pigmentation in the metastatic submaxillary node.

The type of cell forming the growth is the important thing in the histologic diagnosis. Sections show masses of large round or polyhedral cells, varying in size, little or no stroma, numerous blood vessels and no apparent connection between the groups of cells and the overlying mucous membrane.

3. The growth of the primary lesion was slow compared with the rapid dissemination of the metastatic tumors. In 1 case it was eighteen months from the removal of the small growth in the nose until the involvement of the submaxillary node, and there was no recurrence of the primary lesion. In the second case it was over two years before metastasis to the submaxillary area occurred.

This is important from the standpoint of treatment. It indicates that a more complete removal of the tumor, with a wide margin of normal tissue, would offer an excellent chance of cure. This is often difficult to carry out in nasal surgery without leaving marked deformity and impairment of function but should be done for this type of tumor even when the lesion is small. Simple and apparently complete removal of the growth, as in the first case, even if followed by irradiation with radium

or roentgen rays, will be of no permanent benefit. Wide exposure and removal by means of an external approach should be carried out as soon as the diagnosis has been confirmed by careful histologic study. Two methods of approach have been found practical and can be applied, particularly to tumors in the anterior part of the nasal cavity. One is lateral rhinotomy, or Moure operation, in which the incision is through the cheek, along the side of the nose, and the other, the Rouge, or Denker, operation, in which the incision is beneath the upper lip.

The Rouge, or Denker, operation was applied in the second case, combined with ligation of the external carotid artery. Whereas ligation of the artery is not considered necessary by many operators, I found it of sufficient help in maintaining a practically dry field to make the extra trouble well worth while.

CONCLUSION

A most careful histologic study is essential before a proper plan of treatment for malignant tumors of the nasal cavity or sinuses can be formulated.

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PNEUMOPERICARDIUM AND PNEUMOMEDIASTINUM IN CASES OF ACUTE OBSTRUCTIVE LARYNGITIS

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A review of medical literature shows comparatively few cases of invasion of the mediastinal space by air incident to acute obstructive laryngitis. Pneumopericardium has never been so demonstrated. The following cases are therefore presented to the attention of otolaryngologists to indicate the relatively frequent occurrence of these previously held rare conditions. Although the complications in these instances were transitory and all patients recovered, the occurrence of these conditions in the field of otolaryngology was deemed sufficiently important to warrant comment.

All cases studied were encountered in the routine treatment of patients admitted to the croup service of Willard Parker Hospital. Being obstructive infections of the upper part of the respiratory tract, the conditions were brought to the attention of the visiting otolaryngologists. All conditions were identified as nonspecific infectious croup; none were of diphtheritic origin. Of 20 tracheotomized patients observed during the past year, 4 have shown the presence of air in the mediastinal structures. Included in this review is 1 case in which tracheotomy was not performed.

There were no distinctive features present in these cases to differentiate them from other cases under observation in which tracheotomy had been performed. Routine auscultation of the heart and lungs showed a characteristic crepitus over the precordial area, and roentgen examinations revealed the presence of air in the mediastinal tissues. This crepitus is described by Dr. Stern, resident physician of Willard Parker Hospital, as a crackling murmur at the left border of the heart, heard best on expiration and simulating the crushing of cellophane in the hand. Because of numerous rales, rhonchi and harsh breath sounds and the coughing and restlessness of these children, it is difficult to recognize this diagnostic sign. Speculation on the rather frequent appearance of this unusual complication within recent months disclosed greatly improved roentgenograms since these conditions became manifest. This improvement was effected by substantially reducing the time of exposure of each plate, thus producing clearer pictures with minimal

distortion due to cardiac and respiratory motion. High voltage in the tube and the small bodies of children made short exposures possible. By the aid of the improved roentgenographic results, together with the able use of the stethoscope, the definite diagnosis in these cases was established.

The cases here presented include 2 cases of pneumopericardium and 3 cases of pneumomediastinum.

PNEUMOPERICARDIUM

In 1904 James [1] collected 38 cases in literature, and since that time various writers have reported isolated cases in which the condition has been encountered in the routine examination of the thorax. Meyer [2] described a diagnostic roentgen finding of a linear shadow along the border of the heart and parallel with it. A similar finding by Imperatori [3] demonstrated the presence of air or gas in the pericardial space in a case of traumatic perforation of the sac by an open safety pin in the esophagus. In the cases on record etiologic factors include trauma from within and without, pericarditis, with infection with the gas bacillus, erosion and perforation of disease processes from neighboring air-containing organs, and radium necrosis. In a case listed by James [1] sore throat and facial erypsipelas were described as the direct cause. There are no cases on record in which this condition has been associated with acute obstructive laryngitis. When the condition is complicated by fluid in the pericardial sac, a characteristic splashing, water wheel sound is heard. In the absence of fluid, physical examination reveals a metallic, crackling, tinkling cardiac sound. In several instances no characteristic cardiac sound was heard, the condition being discovered on autopsy. Shifting dulness and tympany on percussion are frequently found.

The following cases are presented:

Case 1.—A boy aged 3 years was admitted to the hospital on Nov. 25, 1937, with marked cyanosis, dyspnea with retractions, aphonia, restlessness and signs of circulatory failure. Direct laryngoscopic examination showed all laryngeal structures markedly congested and edematous. Intubation was done pending preparation for tracheotomy. Physical examination at this time showed few breath sounds, no heart murmurs and a temperature of 102.6 F. An emergency tracheotomy was done, and on the following day a shower of crackling rales along the left border of the sternum was noted. A roentgenogram taken at this time disclosed a pneumopericardium (fig. 1 *A*). The laryngeal culture was reported to contain Streptococcus viridans. On this day the temperature rose to 103 F.,

1. James, W. B.: Pneumopericardium, Tr. A. Am. Physicians **19**:351, 1904.
2. Meyer, A.: A Case of Spontaneous Pyopneumothorax Complicated by Hydro- or Pyopneumopericardium, M. Rec. **88**:991, 1915.
3. Imperatori, C. J.: Pericardial-Esophageal Fistula Following a Foreign Body in the Esophagus, Laryngoscope **38**:268, 1928.

and dyspnea continued, coincident with abundant secretions expelled from the tracheotomy tube. A roentgenogram (fig. 1 *C*) taken on the next day (November 27) still showed the characteristic linear shadow parallel with the left border of the heart, not as marked as on the previous day. The lateral view shows a localized substernal collection of air (fig. 1 *B*). The condition of the patient improved under routine treatment, dyspnea became slight, and the friction rub, crackling murmur was less noticeable. A roentgenogram taken on November 29, four days

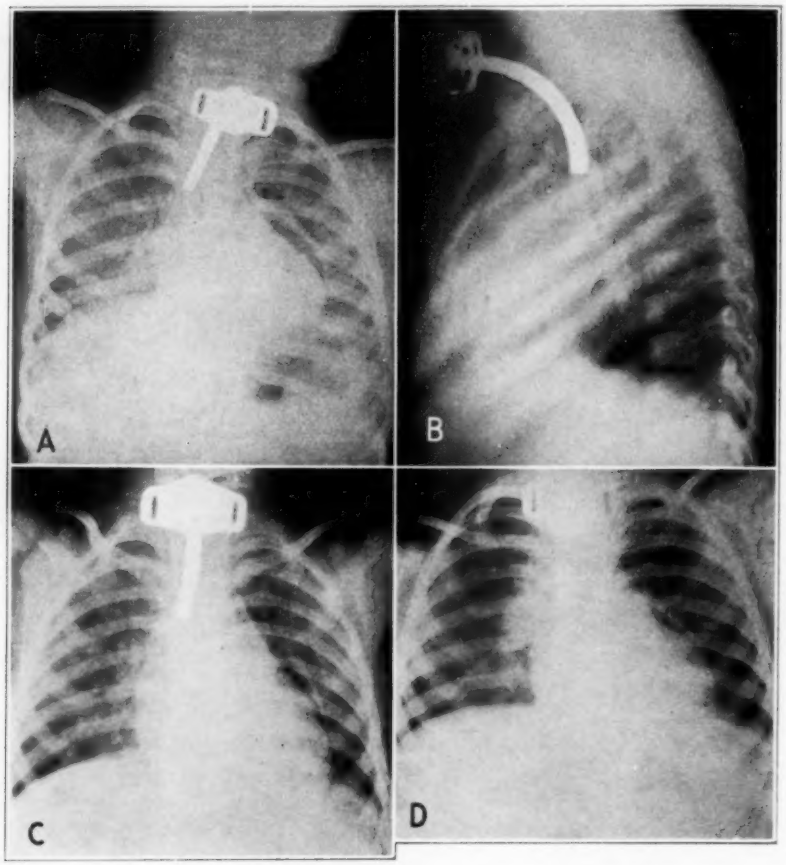

Fig. 1 (case 1).—Roentgenograms of patient.

after admission and three days after the initial positive findings, showed the pericardial air practically absorbed (fig. 1 *D*). The tracheotomy tube was removed on December 3, and roentgenograms taken on this day disclosed complete resolution of all abnormal conditions. The patient was discharged on December 16, twenty-one days after admission to the hospital.

In this case pneumopericardium developed twenty-four hours after tracheotomy. A thoracic examination on the operating table did not disclose any physical findings of mediastinal air. The series of roentgeno-

grams in this case showed the rapid absorption of air and the transitory nature of the condition encountered in all the cases of this review.

CASE 2.—A boy aged 7 years was admitted to the hospital on Dec. 1, 1937, with acute tonsillitis, marked congestion and edema of all supraglottic structures, some respiratory difficulty and a temperature of 102 F. His heart and lungs were normal. He was intubated the following day by the direct method, as respiratory embarrassment was more pronounced and breath sounds were faint at the apexes and absent in the lower lobes. The patient was comfortable while the intubation tube was in place but showed marked respiratory obstruction whenever autoextubation took place. Laryngeal culture was reported to reveal Streptococcus haemolyticus. On December 6 trial extubation was unsuccessful, and on December 7 difficulty in retaining the tube made tracheotomy necessary. The following day the temperature rose; coughing and expulsion of abundant secretions from the tracheotomy tube, retractions and restlessness occurred. Removal of bronchial mucous plugs by suction gave relief and had to be repeated at frequent intervals to keep the airway clear. A roentgenogram taken on this day disclosed the characteristic linear shadow of a pneumopericardium (fig. 2). Many coarse rales, harsh breath sounds and rhonchi were present in both lungs, and the characteristic crepitus along the left border of the sternum was noted at this time. The patient improved after recession of the bronchial reaction and reduction of secretions, and all abnormal cardiac and pulmonary findings on physical examination and roentgenograms had disappeared on December 13. The tracheotomy tube was removed on December 19 and the patient discharged on December 23, twenty-three days after admission.

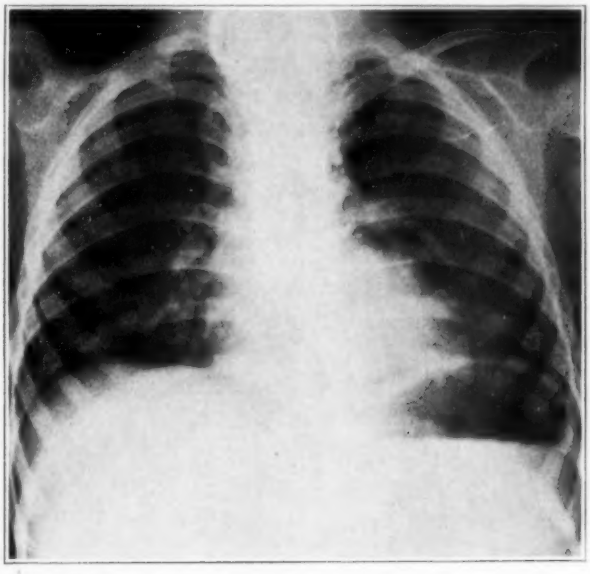

Fig. 2 (case 2).—Roentgenogram of patient.

In this case pneumopericardium and mediastinal air became manifest on the development of respiratory distress incident to bronchial mucous plugs twenty-four hours after tracheotomy was performed.

PNEUMOMEDIASTINUM

Pneumomediastinum is of relatively more frequent occurrence. The symptoms and physical findings closely parallel those of pneumopericardium without fluid. Here again are the characteristic crackling murmur over the precordial area and the abnormal roentgen findings. An additional noteworthy symptom is the presence of pain of a characteristic nature radiating to the neck and axilla from a point behind the sternum, where it took origin. The roentgenograms, however, do not show the diagnostic linear shadow of a pneumopericardium, but disclose the radiolucence of air in place of detail of mediastinal tissue. Among etiologic factors are included severe physical strain of strenuous athletics, described by Scott,[4] causing spontaneous occurrences of the condition, attacks of coughing in bronchopneumonia and whooping cough and, in the majority of cases, pulmonary tuberculosis. Surgical procedures on the neck, thorax, abdomen and tonsils and perforating wounds from within and without have resulted in the occurrence of this condition. Macklin,[5] in his experiments on living animals, was able to produce pneumomediastinum by insufflation of a lobe of a lung, the excessive positive air pressure causing extravasation of air along the perivascular sheaths of the pulmonic vessels into the mediastinum. Continued insufflation eventually resulted in rupture of the mediastinal wall at the point of least resistance, discharge of air into the pleural cavity and massive collapse of the lung. LeWald [6] expressed the opinion that spontaneous pneumothorax may occur in a person with healthy lungs and result in complete and permanent recovery and that air may pass from one pleural cavity to the other, thus producing bilateral pneumothorax. Whether the condition is secondary to increasing primary pneumomediastinum or is directly due to rupture of the visceral or parietal pleura cannot always be determined. Macklin [5] found in his experiments that air will escape readily along the perivascular sheaths of the pulmonic vessels, and for this reason he was unable to inflate a normal lung sufficiently to produce rupture of the pleura. In the cases here presented there was a moderate transitory pneumomediastinum with no evidence of pneumothorax.

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4. Scott, A. M.: The Significance of the Anginal Syndrome in Acute Spontaneous Pneumomediastinum, Lancet **1**:1327, 1937.

5. Macklin, C. C.: Pneumothorax with Massive Collapse from Experimental Over-Inflation of the Lung Substance, Canad. M. A. J. **36**:414, 1937.

6. LeWald, L. T.: Bilateral and Unilateral Nontuberculous Spontaneous Pneumothorax, Arch. Surg. **12**:440 (Jan., pt. 2) 1926.

CASE 3.—A boy aged 7 years was admitted to the hospital on Jan. 4, 1938, with findings of measles, pertussis and acute obstructive laryngitis. Direct laryngoscopic examination showed marked congestion of the pharynx and edema of the epiglottis and aryepiglottic folds. Cardiac sounds were faint, and a systolic murmur was heard at the left border of the sternum. There was moderate substernal pain radiating to the neck. Dyspnea and retractions became more pronounced, and tracheotomy was done. In separating the deep structures overlying the trachea, air bubbles were seen to escape, and miid subcutaneous emphysema of the neck developed during completion of the operation. Insertion of the tracheotomy tube gave relief of the respiratory distress, and the patient did not have any further trouble from this source. A roentgenogram taken on the follow-

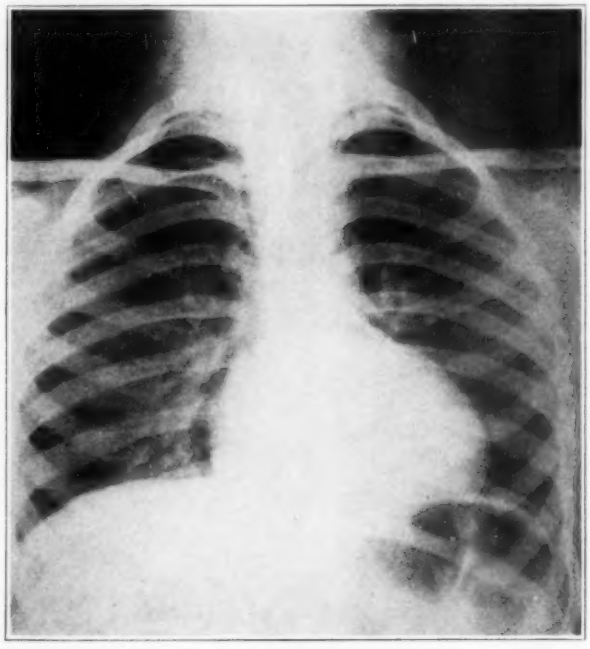

Fig. 3 (case 3).—Roentgenogram of patient.

ing day showed the presence of pneumomediastinum (fig. 3), and a crackling murmur at the left border of the heart was clearly audible. The laryngeal culture showed Staphylococcus aureus. During the following days the patient improved rapidly; daily roentgenograms showed gradual absorption of mediastinal air, and on January 13 all findings were essentially negative. The tracheotomy tube was removed on January 18, and the patient was discharged on January 23, seventeen days after admission.

In this case pneumomediastinum apparently preceded tracheotomy, escape of air being noted during operation. A systolic murmur and faint cardiac sounds were noted prior to operation, and the characteristic crepitus was present twelve hours later.

CASE 4.—A girl aged 16 was admitted to the hospital on June 7, 1937, with sore throat, listlessness, difficulty in swallowing and a temperature of 104 F. Direct laryngoscopic examination disclosed marked congestion of the epiglottis but no dyspnea or retractions; the airway was sufficiently clear for respiration. Cardiac and pulmonary sounds were normal. The following day the patient had severe pain in the throat, was unable to swallow food and had a temperature of 104.8 F., a moderate systolic murmur over the third and the fourth interspaces and a croupy cough, with expectoration. Pain in the throat became more marked, radiating from beneath the sternum. A loud friction rub, crackling murmur over the precordial area was heard, more marked on expiration. A roentgenogram taken

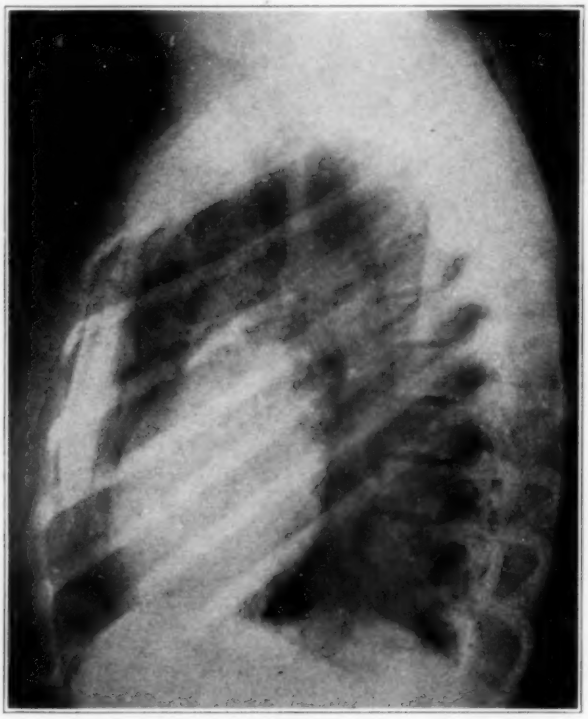

Fig. 4 (case 4).—Roentgenogram of patient.

on this day disclosed pneumomediastinum (fig. 4). Laryngeal culture was reported to reveal Str. haemolyticus. Two days later the precordial crepitus had disappeared; routine roentgenograms on subsequent days showed gradual absorption of mediastinal air, physical and roentgen examination on June 16 disclosing a normal heart and lungs.

In this case tracheotomy was not indicated. However, on June 9, when a cough with expectoration developed and the patient showed other evidence of infectious involvement of the lower part of the respiratory tract, pneumomediastinum was found.

CASE 5.—A boy aged 2½ years was admitted to the hospital on Feb. 15, 1938, with a diagnosis of acute laryngitis. Direct laryngoscopic examination showed general congestion of the larynx, moderate subglottic edema, mucopus expelled from the subglottic region and slight dyspnea and retractions. Laryngeal suction and intubation by the direct method were done the following day for relief of increasing dyspnea. The laryngeal culture showed Str. viridans. There were some physical findings indicative of beginning tracheobronchitis and bronchopneumonia on February 18. Roentgenograms taken during the following week showed no abnormality. The patient was comfortable as long as the intubation tube was in place, but repeated trial extubations were unsuccessful. Because of erosion of the larynx, tracheotomy was performed on February 26. Two days later symptoms of marked bronchopneumonia were noted; the temperature rose to

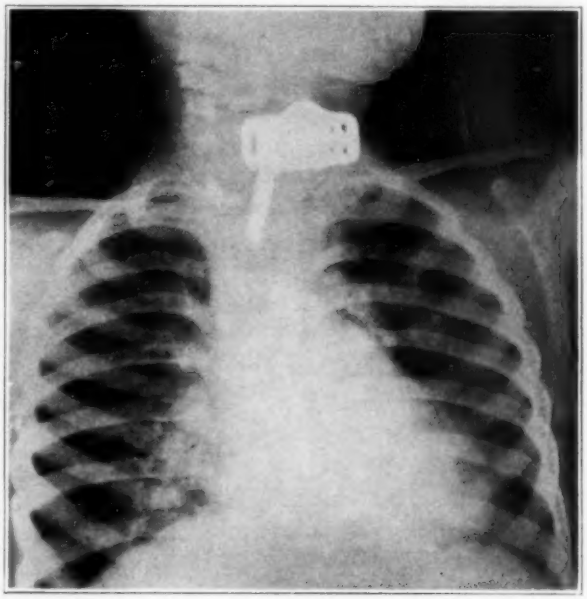

Fig. 5 (case 5).—Roentgenogram of patient.

105.2 F.; abundant secretions were expelled from the tracheotomy tube, and ausculation showed numerous crackling rales along the left border of the sternum. A roentgenogram taken at this time showed a considerable collection of air beneath the mediastinal pleura (fig. 5). Substernal tenderness was present on pressure. The patient's convalescence was slow; frequent suction through the tracheotomy tube was followed by relief of respiratory embarrassment and a drop in temperature. Although physical examination continued to show an active bronchial infection, the characteristic crepitus along the left border of the sternum lessened and was absent on March 2. Roentgenograms taken on March 7 showed complete absorption of mediastinal air. The tracheotomy tube was removed on March 21.

In this case pneumomediastinum developed two days after tracheotomy and at the time of a marked activation of previously mild broncho-

pneumonia. Relief of respiratory embarrassment by frequent suction of the bronchi was followed by prompt absorption of mediastinal air.

COMMENT

The question raised by these cases is by what route invasion of the mediastinum by air took place. Was air sucked in through the tracheotomy wound by the inspiratory efforts and the normal negative pressure in the mediastinum, or was air forced out of the substance of the lung and along the perivascular sheaths into the mediastinum by excess positive pressure incident to respiratory obstructions? The preponderance of evidence in the cases here presented would indicate a direct relation of these complications to obstruction of the upper and the lower part of the respiratory tract rather than to the performance of tracheotomy with its attendant wound.

In case 4 tracheotomy was not performed, but a well defined pneumomediastinum developed.

In case 3 bubbles of air were seen to escape on separation of the ribbon muscles overlying the trachea. Apparently there was sufficient positive air pressure in the mediastinum to produce the phenomenon on separation of restraining tissues.

In case 2, in which elective tracheotomy was done, pneumopericardium developed twenty-four hours after tracheotomy and the sealing of the wound by a snugly fitting petrolatum gauze pack around the tube. The complication became manifest at a time of respiratory distress due to bronchial mucous plugs and was not present immediately after operation.

In case 1 pneumopericardium developed twenty-four hours after tracheotomy and the sealing of the wound. Clinical findings indicated infection of the lower part of the respiratory tract at the time of occurrence.

In case 5 a similar process took place forty-eight hours after operation and at a time when bronchopneumonia with abundant secretions caused respiratory embarrassment.

In reference to the occurrence of pneumopericardium, how air entered the pericardial sac is unknown. Both patients recovered, and the question could not be solved by autopsy. It is felt, however, in accord with the experimental production of pneumomediastinum in living animals by Macklin,[5] that air found its way along the perivascular sheaths of pulmonic vessels into the mediastinum and gained entrance into the sac where the pericardium is deflected along these vessels. On inquiry relative to this question, Dr. Macklin replied that he had never seen evidence of pneumopericardium in any of his experimental animals. Careful search of the literature fails to disclose the

point of entrance of air in a case of spontaneous pneumopericardium or that the condition has been produced experimentally in living animals.

TREATMENT

Extrapulmonary air required no specific treatment in these cases beyond the control of the flow of air into the mediastinum by removal of obstruction to respiration. The occurrence of this complication did not influence the prognosis directly, and it never played a conspicuous part in the clinical picture. The air was quickly absorbed after its continued extravasation was stopped by elimination of obstructions. Neffson and Wishik [7] described in detail the routine treatment of 727 patients with infectious croup admitted to Willard Parker Hospital, the medical and operative treatment set forth being followed today. To eliminate as far as possible the formation of obstructive crusts and mucous plugs, constant steam inhalation in enclosed cubicles is prescribed for all tracheotomized patients. Special nursing care and frequent suction of secretions through the tracheotomy tube after instillation of small quantities of 5 per cent solution of sodium bicarbonate keep the airway clear of tenacious mucus and crusts. Bronchial plugs are removed by the bronchoscope and Tolle suction tubes passed through the tracheotomy wound. Control of the infection of the upper part of the respiratory tract is effected by topical applications. Sedatives and atropine are never used. Supportive measures are instituted on signs of circulatory failure.

Kernan and Barach [8] reported favorable results in a large percentage of cases in which helium and oxygen were inhaled by means of a hood tent. Their clinical results indicate a marked reduction of respiratory embarrassment and less need of surgical intervention.

It is the policy of Willard Parker Hospital to decannulate tracheotomized patients as soon as feasible. Gradual plugging of the tube as soon as decreased laryngeal obstruction permits an adequate airway is quickly followed by removal of the tube if no respiratory difficulty ensues. In the cases here presented the numbers of days of cannulation were as follows: case 1, nine days; case 2, thirteen days; case 3, thirteen days; case 4, no tracheotomy; case 5, twenty-three days.

CONCLUSIONS

1. Pneumopericardium and pneumomediastinum may occur at any time in the course of acute obstructive laryngitis. Their occurrence is

7. Neffson, A. H., and Wishik, S. M.: Acute Infectious Croup: General Study of Acute Obstructive Infections of the Larynx, Trachea, and Bronchi with Analysis of Seven Hundred and Twenty-Seven Cases, J. Pediat. **5**:433, 617 and 776, 1934.

8. Kernan, J. D., and Barach, A. L.: Role of Helium in Cases of Obstructive Lesions in the Trachea and Larynx, Arch. Otolaryng. **26**:419 (Oct.) 1937.

directly influenced by obstruction of the upper and the lower part of the respiratory tract.

2. The complication is not directly due to or proximately the result of tracheotomy per se. It occurred in a case in which tracheotomy was not performed; it was apparently present prior to operation in another, and sealing the tracheotomy wound against air in 3 cases did not prevent its occurrence twenty-four to forty-eight hours later.

3. The conditions do not play a conspicuous part or influence the course or prognosis of the disease with which they are associated.

4. Pneumopericardium and pneumomediastinum are of relatively frequent occurrence. Repeated roentgen and physical examinations at short intervals will probably reveal positive examples that may easily be overlooked.

5. The mode of production of spontaneous pneumopericardium is unknown.

The radiology department of Willard Parker Hospital made the roentgenologic observations and permitted the use of their records. Dr. Morris E. Stern, resident physician, cooperated in the assembling of these data.

45 Park Avenue.

TUMORS OF THE NOSE AND THROAT

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Portmann and two collaborators [1] made a detailed study of the histologic structure in 100 cases of carcinoma of the upper air and food passages in an effort to determine the importance of the microscopic appearance of the tumors in the prognosis and choice of treatment. This study included the structure of the growths (degree of differentiation, frequency of mitotic figures) and the reaction of the surrounding tissues. The authors concluded, especially on the basis of the slow evolution of carcinomas of the larynx, of which there were 61 in their series, that the histologic picture frequently does not correspond to the clinical course of the disease and must not influence the clinician too strongly.

Goldsmith and Ireland [2] reported 6 aberrant mixed tumors of salivary gland type; 4 of these tumors were in the soft palate, 1 in the antrum and 1 in the nasopharynx and palate. They expressed the opinion that this type of tumor is not a true teratoma, and they agreed with Ewing that it represents a fully formed glandular tissue which has undergone change. They advocated surgical removal followed by irradiation. They obtained poor results in 3 cases in which they used only irradiation therapy.

NOSE

Weidlein [3] reported a case in which a mixed tumor of the salivary gland type arose from the left side of the nasal septum of a woman aged 53. He found only 1 other report of a similar case in the literature. Two good photomicrographs were included in his report.

Sprowl [4] briefly reviewed the literature on osteomas of the nose and reported a case in which an osteoma arose from the left side of the roof

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* Member of the Section on Laryngology, Oral and Plastic Surgery.

1. Portmann, G.; Barraud, A., and Mougneau: Des rapports de la malignité histologique et de la malignité clinique de l'épithélioma des voies aéro digestives supèrieures, Acta oto-laryng. **24**:135-150, 1936.

2. Goldsmith, P. G., and Ireland, P. E.: Mixed Tumors in the Nose and Throat, Ann. Otol., Rhin. & Laryng. **45**:940-950 (Dec.) 1936.

3. Weidlein, I. F.: Mixed Tumor of the Nasal Septum: Report of a Case, Ann. Otol., Rhin. & Laryng. **45**:574-577 (June) 1936.

4. Sprowl, F. G.: Orbito-Ethmoidal Osteoma with a Report of the Case, Ann. Otol., Rhin. & Laryng. **45**:886-890 (Sept.) 1936.

of the anterior ethmoid labyrinth of a man aged 18. Osteomas of the nasal accessory sinuses are uncommon. Those arising from the ethmoid bone are especially rare and usually develop at the junction of the orbital plate with the ethmoid bone. Their causation is uncertain. Trauma has been considered a factor, as has chronic infection. These tumors are of three types: eburnated, spongiose and mixed. It is important but difficult to distinguish them from exostoses, which have no tendency to progress and may be treated by simple removal of the thickened portion. Osteomas are true neoplasms; they are capable of unlimited growth and must be removed in toto, with the base, to prevent recurrence. Early removal is advisable to prevent extension to vital regions.

Thirty-five cases of malignant tumor of the nose and its accessory sinuses, which were observed at the Groningen Otorhinolaryngologic Clinic during the course of twelve years and reported elsewhere, formed the basis of a discussion of this subject by Huizinga.[5] In 12 of the cases, there was no tumor in the nasal cavity and the main symptom was a swelling of the upper jaw or of the internal canthus of the eye. The author discussed the relative difficulty of diagnosis by roentgen rays, especially the differential diagnosis of these tumors, mucocele and pyocele. The use of a large aspirating needle was found to provide sufficient tissue for biopsy in most cases. Tumors of the frontal sinus were found to be especially difficult to diagnose. Twenty-five patients were found to have carcinoma, and the remaining 10 had sarcoma. This rather high percentage of sarcoma arouses the thought that highly malignant and undifferentiated carcinomas may have been confused with sarcomas. The author discussed the pathologic changes in some cases at great length, especially the possibility that some of the tumors may have been carcinosarcomas. The photomicrographs and discussions were not convincing on this point.

Hall [6] reported 9 cases of true papilloma of the nose observed at the Sabbatsberg Clinic between 1918 and 1936. In general, he arrived at the conclusions reached by Kramer and Som [7] in their recent report. The tumors concerned, which are comparatively rare (about 70 have been reported), were described by the author as hard papillomas, in distinction from the soft or mucous type. They are inflammatory and rarely invade the sinuses, although such involvement occurred in 2 of the authors' cases. Rarely such tumors are primary and limited to the

5. Huizinga, E.: Diagnostische Schwierigkeiten bei malignen Nasennebenhöhlentumoren, Acta oto-laryng. **25**:296-310, 1937.

6. Hall, A.: Contribution à l'étude clinique du papillome dur des fosses nasales, Acta oto-laryng. **25**:226-239, 1937.

7. Kramer, R., and Som, M. L.: True Papilloma of the Nasal Cavity, Arch. Otolaryng. **22**:22-43 (July) 1935.

sinuses. They are more frequent among men than among women and usually occur in the fourth decade of life. A differential diagnosis depends on the microscopic examination, which will show the characteristic fibrous stroma and thick epithelium. In the mucous type plasma cells are present in great number. The authors divided these growths into cylindric, squamous and mixed types. While they are not primarily malignant, they have a great tendency to recur, either because of incomplete removal or because they have a tendency to form elsewhere in the mucosa. Incomplete removal appears to increase the rapidity of growth. The authors found that irradiation alone had little or no effect on this type of growth, while a combination of diathermy and irradiation produced good results. They found that it was unnecessary, even in cases of papilloma arising in the posterior part of the nose, to employ any but the intranasal approach, which avoided many of the unpleasant consequences of a more radical approach. These tumors, which produce no symptoms except a gradually increasing obstruction, should be removed as early as possible because of their tendency to become malignant.

NASOPHARYNX

Gerlings and den Hoed [8] reported 47 cases of malignant tumor of the nasopharynx in which the patients were treated at the Antoni Van Leeuwenhoekhuis Cancer Clinic in Amsterdam between 1915 and 1935. The authors stressed the necessity of early diagnosis, which is difficult because of the inaccessibility of the region concerned. They reviewed the available methods of examination, which are anterior and posterior rhinoscopy with Yankauer's speculum and the nasopharyngoscope, palpation, roentgenography after injection of iodized poppyseed oil, biopsy after removal of a specimen with the diathermic knife to avoid hemorrhage and complete examination of the blood to exclude the possibility of a lymphatic disease. They then briefly, but in detail, reviewed the various symptoms and symptom complexes characteristic of this disease. It is now recognized that the only treatment is irradiation. Operation is both dangerous and futile; it also is accompanied by almost insurmountable technical difficulties. Tables which were included showed the age and sex of the patients, the type of growth and the results of treatment. From these tables it appears that the male patients outnumbered the female patients by 2 to 1, that there were a large number of young patients (9 under 20 and 27 under 50 years of age). There were 4 epidermoid carcinomas, 16 anepidermoid carcinomas (of these only 3 were lymphoepitheliomas) and 14 sarcomas. The treatment was varied continuously through the years; the most recent treatment consisted of a

8. Gerlings, P. G., and den Hoed, D.: Diagnostik und Therapie der bösartigen Nasenrachengeschwülste, Ztschr. f. Hals-, Nasen- u. Ohrenh. **41**:385-398, 1937.

modified Coutard method. It was noteworthy that the results did not appear to change much with the change of method. The best results were obtained in cases of sarcoma, and the next best results were obtained in cases of lymphocarcinoma. The authors attributed this to the greater sensitivity of these tumors to irradiation. In 8 cases, or 17 per cent, the patients were well from one and a half to nineteen years. In 6 of these cases the tumors were sarcomas. Radium was inserted directly into the nasopharynx; this was combined with roentgen therapy in the later years. The treatment must be thorough the first time, as recurrences are little affected by further treatment. The authors expressed the opinion that earlier diagnosis and further improvement in treatment offer great hopes for the future.

Schlivek [9] reviewed 38 cases of malignant tumor of the nasopharynx observed at Mount Sinai Hospital in New York and reported in detail 16 of these cases in which symptoms were referable to the eye. These symptoms were as follows: involvement of the trigeminal nerve in 12 cases, Horner's syndrome in 10 cases, involvement of the sixth cranial nerve in 8 cases, involvement of the seventh cranial nerve in 5 cases, ophthalmoplegia interna and externa in 4 cases, exophthalmos in 3 cases, ptosis in 2 cases, papillitis in 1 case, atrophy of the optic nerve in 1 case and a mass in the orbit in 1 case. A sphenoid foramen syndrome, that is, paralysis of the third, fourth and sixth cranial nerves and the ophthalmic branch of the fifth cranial nerve, was present in 2 cases. The author reviewed the other symptoms and syndromes produced by malignant growths in this region and discussed the frequent errors of diagnosis and the mistakes they caused in treatment, and he stressed the importance of an accurate examination of the nasopharynx in cases in which symptoms were referable to the cranial nerves. He discussed the mechanism by which the Horner syndrome is produced and expressed the opinion that it may be due to involvement of post-ganglionic fibers returning to the skull from cervical ganglions. He suggested that a syndrome which involves the fifth cranial nerve, the sympathetic nerves and the sixth cranial nerve and is associated with symptoms referable to the ear may be called an ocular syndrome of nasopharyngeal tumor. In the discussion it was suggested that the Horner syndrome was due in many cases to pressure on the so-called paratrigeminal portion of the sympathetic nervous system, which accompanies the trigeminal nerve.

Koch and Eigler,[10] in reporting 6 cases of nasopharyngeal fibroma, advocated the use of irradiation in the treatment of this disease, especially

9. Schlivek, K.: Ocular Manifestations of Malignant Nasopharyngeal Tumors: Report of Cases, Arch. Ophth. **17**:1055-1069 (June) 1937.

10. Koch, J., and Eigler, G.: Beiträge zur Strahlenbehandlung der Basalfibroide, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **142**:1-14 (Oct.) 1936.

when the tumor is extensive and invades the nose and sinuses. In such cases surgical treatment is dangerous because of the tendency to uncontrollable hemorrhage. The mortality of operative treatment is high (between 10 and 20 per cent), and the after-results frequently are mutilating. Atrophic conditions also may make life miserable for the patient in later years. In all the authors' cases the patients responded to radium and roentgen therapy, especially to the former. No unpleasant after-effects were noted. Coutard's method of roentgen therapy was used in cases in which the involvement was extensive, and the results were satisfactory. The authors varied the dose according to the age of the patient and the extent of the tumor. They were unable to make any distinction in their treatment from the histologic appearance of the tumor, which varied in all cases and in different portions of the tumors which were removed for biopsy.

Bazzana [11] reported 19 cases of nasopharyngeal malignant tumor observed in the City Hospital of Brescia in five years. The article included a description of the anatomy of the nasopharynx and the neighboring regions. It also included a description of the pathology of the various forms of malignant growths of the nasopharynx, thorough description of the various clinical forms of this disease and a description of the various cranial nerve syndromes. The author stressed the importance of roentgen examination in these cases, both for diagnostic purposes and to follow the extension of the tumor along the base of the skull. In 8 of the cases the tumors were sarcomas; in 6 cases they were reticulomas, and in the remaining cases they were squamous cell carcinomas.

PHARYNX

One hundred and forty-seven patients who had malignant tumors of the tonsil were treated between 1916 and 1932 at the Van Leeuwenhoekhuis Clinic in Amsterdam. Levie [12] reviewed these cases briefly as regards the classification and treatment of the tumors. He found that the cases could be divided into four clinical groups, in which there were definite differences in symptoms and course; in almost 80 per cent of the cases the symptoms and clinical course corresponded with the histologic structure of the tumor. The first group consisted of cases of epidermoid carcinoma of both cutaneous and mucous type; however, in cases in which the tumor was of the mucous type there was some tendency to keratinization. The second group included cases of various

11. Bazzana, E.: I tumori maligni del rinofaringe: Considerazioni cliniche, radiografiche, röntgen e curieterapiche su 19 casi, Oto-rino-laring. ital. **6**:319-402 (Aug.) 1936.

12. Levie, B.: Classification and Treatment of Malignant Tumors of the Tonsils, Acta oto-laryng. **25**:403-413, 1937.

types of nonkeratinizing squamous cell carcinomas. The third group consisted of transitional cell tumors and lymphoepitheliomas (which the author considered one and the same). The fourth consisted of sarcomas, mainly lymphosarcomas and round cell sarcomas. The author found that in the first two groups tobacco, alcohol and syphilis appeared to be definite etiologic factors, while they were unimportant in the other two groups. One hundred and seventeen of the patients were men, and 30 were women. Tables showed the various groups of tumors and the result with different methods of treatment. In cases in which the tumors were sarcomas or lymphoepitheliomas the best results were obtained with pure roentgen therapy, and in cases in which epidermoid tumors were encountered the best results were obtained with a combination of diathermy, radium and postoperative roentgen therapy. Metastatic involvement was present in about 70 per cent of the cases; therefore, the results were not encouraging. Thirteen patients lived more than five years; all of these but 1, who survived seventeen years, died of recurrence or generalization of the malignant growth. Coutard's method is now being used, but it has not been used long enough to permit an evaluation of the results.

Pilcher [13] reported 100 consecutive cases of carcinoma of the pharynx observed at the University College Hospital in the years 1926 to 1935 inclusive. The tumors were classified, according to their site of origin, as epilaryngeal and postcricoid or hypopharyngeal. Epilaryngeal tumors include those which arise from the epiglottis, the aryepiglottic fold, the lateral wall of the pharynx or the piriform sinus. In the 71 cases of epilaryngeal tumor 70 of the patients were males and 1 was a female. In the 29 cases of postcricoid tumor all the patients were females. Postcricoid or hypopharyngeal tumors have a tendency to occur about ten years earlier in life than do epilaryngeal tumors. The author said that apparently trivial symptoms, such as a sense of fulness in the throat, tickling and slight pain, should not be dismissed lightly. The early symptoms of this condition are always trivial and should always warrant an accurate laryngoscopic examination, and if doubt still exists an esophagoscopic examination should be performed. Indirect laryngoscopic examination will almost always reveal actual lesions, fulness and alteration in the movement of the arytenoid cartilages or a collection of mucus in the piriform fossa. The appearance of dysphagia, hoarseness or palpable lymph nodes is evidence of an advanced growth. Surgical treatment by lateral pharyngotomy offers some chance of life; the value of irradiation in this series of cases has not been determined.

13. Pilcher, R.: Diagnosis of Malignant Disease of the Pharynx, Brit. M. J. **1**:13-16 (Jan. 2) 1937.

The author reported the results of surgical treatment in 100 cases. The lesion was operable in 49 cases; in 35 of these cases the patients were males, and in 14 cases they were females. Radical operations were completed in 39 cases. In these cases 25 patients were males, and 14 were females. Among the 25 males the operative mortality was 9. Four of the males were alive at the time of the author's report. Among the 14 females the operative mortality was 7. Three of the females were alive at the time of the author's report.

Richards [14] reported in detail 52 cases of malignant tumor involving the tonsil and its pillars. These cases were observed in the years 1928 to 1935, inclusive, at the Institute of Radiotherapy of Toronto General Hospital. Of these patients 42 suffered from carcinoma, predominantly of the epidermoid type, and 10 suffered from various forms of sarcoma. The cases and the results obtained were thoroughly analyzed. Although the number of cases was not large and the number of years which had elapsed since treatment was too small to warrant any conclusions, the results obtained, especially as to primary healing, appeared promising. The author expressed the opinion that the radium bomb offers the best results in this type of lesion.

Schinz and Zuppinger [15] published an extensive and detailed account of the treatment of 190 patients who had malignant tumors of the mesopharynx, including the tonsil, the angle between the pillar of the tonsil and the tongue, the base of the tongue, the valleculae and the lateral and posterior walls of the pharynx. There were 14 sarcomas of the tonsil, 45 carcinomas of the tonsil, 18 carcinomas of the angle between the pillar and the tongue, 39 carcinomas of the base of the tongue, 29 carcinomas of the valleculae, 10 carcinomas of the lateral wall and 8 carcinomas of the posterior wall. In 8 cases of sarcoma of the tonsil, in 6 cases of carcinoma of the tonsil and in 3 cases of carcinoma of the base of the tongue, the patients were women. The authors compared the results obtained in these cases, in which the patients were treated at the Zurich University Roentgeninstitut between 1929 and 1933, with those obtained in a smaller group of patients who were treated between 1918 and 1928. In the earlier group various methods of treatment were used; in the later group Coutard's protracted fractional [16] method was used exclusively. The method of classification of cases, that is, as to the type and extent of the lesion and the presence of metastasis, is rather

14. Richards, G. E.: The Radiological Treatment of Cancer; Methods and Results, 1928-1935: III. Malignant Lesions of the Tonsil and Its Pillars, Canad. M. A. J. **35**:385-390 (Oct.) 1936.

15. Schinz, H. R., and Zuppinger, A.: Zürcher Erfahrungen der Radiotherapie von bösartigen Mesopharynxgeschwülsten, Arch. f. klin. Chir. **187**:582-613, 1936.

16. Radium needles and electrocoagulation were used in some cases to destroy tumor rests.

cumbersome and complicated, but the great strides achieved are evident. In spite of the group's comprising all stages of malignant disease and in the greatest number of cases hopelessly advanced growths, a temporary disappearance of the primary and regional lesions was obtained in 52 per cent of cases and palliative improvement in almost all. These figures are even better if only those patients who underwent a complete course of treatment are considered. Twenty-four per cent remained free of symptoms for three years and 15 per cent for five years. These figures climb respectively to 37.5 per cent and 24 per cent if the patients who did not complete treatment are excluded. There is still apparent some confusion in the nomenclature of the different types of tumors, among both the sarcomas and the carcinomas, which it would be desirable to eliminate.

Ducuing and Ducuing [17] advocated that malignant tumors which arise in the "cricopharyngeal belt" should be recognized as a separate clinical entity. This distinction was suggested by Zuppinger in 1931. According to these authors, the cricopharyngeal belt extends from the upper level of the cricoid cartilage to its lower margin and from one piriform fossa to the other. It appears to be the same as that more loosely known in the literature as the "postcricoid region." Tumors of this region usually produce symptoms earlier than do those of the neighboring piriform fossa; they are characterized by their prevalence among women. The early symptoms are palpable lymph nodes in the jugular region and disturbances of deglutition which frequently are accompanied by pain. Late symptoms, especially if the tumor arises from the cricoid cartilage, involve respiratory obstruction. Treatment consists in the various forms of irradiation; operation for obvious reasons is limited to emergency procedures directed to the relief of respiratory obstruction and interference with deglutition. Although the mortality rate is 100 per cent, the Coutard method offers the best results. The authors employed this treatment in 7 cases; all the patients survived less than six months.

LARYNX

Buckley [18] discussed carcinoma of the larynx and its treatment in detail. He felt that as yet roentgen therapy and radium therapy do not produce the consistently good results that operation does. He described his technic. Chief interest of the article lies in the author's practice of removing the hyoid bone completely. This insures adequate exposure during the operation and permits easy and firm closure of the esophageal

17. Ducuing, J., and Ducuing, L.: Les tumeurs malignes de la "ceinture" cricopharyngée, Rev. de laryng. **57**:1085-1105 (Dec.) 1936.

18. Buckley, R. E.: Carcinoma of the Larynx and Its Management, Tr. Am. Laryng. A. **58**:132-143, 1936.

wound. To obviate bleeding from the tracheal stump the author injects epinephrine into the tracheal mucosa before severing the posterior wall of the trachea.

Looper [19] reported 35 cases of laryngectomy in which there was no operative mortality. He described his technic, which consists of a one stage operation without a preliminary tracheotomy. He advocated a semilunar flap, for the purpose of diminishing the chance of a postoperative esophageal fistula. He placed great emphasis on hemostasis and avoidance of contamination from the mouth. The chief variant in his method is the insertion, before the esophagus is opened, of mattress sutures all around its attachment to the larynx. These sutures later are used to invert the margins of the pharyngeal mucosa and firmly close the esophageal opening. He also leaves a tongue-shaped flap on the posterior wall of the tracheal stump, which facilitates its attachment to the skin and protects the esophageal wall when a tracheal tube is used. The author said that at present operation is still the method of choice in the treatment of tumors in this region.

Patterson [20] described a method for the conservative surgical treatment of malignant growths involving the anterior commissure of the larynx and a portion of both vocal cords. He used this method in 7 selected cases, in which he felt that entire extirpation of the larynx was unwarranted and thyrotomy insufficient. The method, which has points of similarity to the methods of Hautant, Portmann and Jackson, consists of exposure of the larynx by means of a flap, which is shaped like a question mark. The thyroid cartilage is denuded without injuring the perichondrium; this procedure is followed by removal of the cartilage with a punch forceps, without penetrating the laryngeal cavity. When the cartilage overlying the growth and its surroundings has been widely removed, a tracheal cannula is inserted, and the larynx then is opened, care being taken to avoid the growth. The affected region is excised. The author advocated the wide use of the endothermic current.

Diggle [21] reviewed carefully and at great length the results obtained in intrinsic laryngeal cancer suitable for laryngofissure or irradiation with the Finzi-Harmer technic by treatment with these two methods. Radium therapy was used in 26 cases, and laryngofissure, in 28 cases. There was no mortality in the group in which irradiation was used, but in the cases in which laryngofissure was performed the operative mortality was 11.1 per cent. Of the patients who were treated with irradia-

19. Looper, E. A.: Laryngectomy for Cancer of the Larynx, Surg., Gynec. & Obst. **64**:524-530 (Feb. 15) 1937.

20. Patterson, N.: Carcinoma of the Larynx: A Plea for More Conservative Surgical Procedures in Certain Cases, Arch. Otolaryng. **23**:295-303 (March) 1936.

21. Diggle, F. H.: The Treatment of Intrinsic Laryngeal Cancer, J. Laryng. & Otol. **52**:463-476 (July) 1937.

tion, 77.7 per cent were free of symptoms for more than three years; 61.1 per cent of the patients who were treated surgically were free of symptoms for a similar period. The tumors recurred in 22.2 per cent of the cases in which irradiation was employed but in only 16.6 per cent of the cases in which the patients were treated surgically. The functional results were "good" in 58 per cent of the cases in which irradiation was used and in 70 per cent of the cases in which the patients were treated surgically. In conclusion, the author said that in spite of the apparent better results as regards period of survival and operative mortality, operative treatment has the advantage. Among other things it was noted that convalescence was much more prolonged in the cases in which irradiation was employed. There was no information as to possible later injury from the radium treatment, as the period which had elapsed since the irradiation was brief. The author also expressed the opinion that the high mortality in the cases in which surgical treatment was employed, which does not agree with the low mortality reported by well known laryngologists, was in part due to the fact that the patients had been operated on by a number of different surgeons, some of whom had had a comparatively limited experience.

Weidman and Schaffer [22] reported an interesting case of extensive xanthomatosis, in which the larynx was involved. The patient, who died, had symptoms of involvement of the central nervous system. At necropsy a xanthomatous tumor was found in the region of the pons. The cause of death appeared however to be a gastric carcinoma. It was brought out during the discussion that in one of the cases reported recently by Finney, Montgomery and New [23] also necropsy disclosed a xanthoma of the pons. This thorough article is well worth reading in its entirety. The connection between xanthoma, diabetes mellitus, cholesterol and fat metabolism is exhaustively discussed.

TRACHEA

Lukens [24] reported a case of primary papilloma of the trachea in which the patient was a boy aged 7 years. The papilloma was repeatedly removed by scraping with the end of the bronchoscope. The main symptom was dyspnea. Only 2 other cases of papilloma limited to the trachea had been observed at the Jefferson Hospital Bronchoscopic Clinic.

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22. Weidman, F. D., and Schaffer, H. W.: Xanthoma of the Skin and Larynx Associated with Carcinoma of the Stomach and a Regressive Xanthoma of the Pons, Arch. Dermat. & Syph. **35**:767-811 (May) 1937.

23. Finney, W. P.; Montgomery, H., and New, G. B.: Xanthoma Multiplex: Two Cases Involving the Larynx and Trachea, and Associated with Diabetes Insipidus, J. A. M. A. **99**:1071-1074 (Sept. 24) 1932.

24. Lukens, R. M.: Papilloma of the Trachea: Report of a Case, Ann. Otol., Rhin. & Laryng. **45**:872-874 (Sept.) 1936.

Guttman [25] reported a case of adenocystic carcinoma of the trachea in which the patient was an Indian woman aged 32, who was suffering from increasing dyspnea. The growth was discovered during a tracheotomy, which was performed to relieve the respiratory difficulty. It was later found to be demonstrable by roentgenography. The author reviewed the literature and discussed the pathologic changes that were present in his case. He concluded that this type of tumor can be said to be a basal cell epithelioma arising from the mucous membrane. He accepted Krompecher's classification. The growth is comparatively benign, like its prototype in the skin, and has little or no tendency to metastasize or ulcerate. He advised high voltage roentgen therapy as the treatment of choice.

ESOPHAGUS

Rose [26] reported a case in which multiple myomas were found at necropsy. The patient was a man aged 74. A diagnosis of carcinoma of the esophagus had been made. This condition appears to be a rare one; the author found only 49 similar cases reported in the literature. The condition usually is discovered at necropsy. In only 6 of the cases were the symptoms referable to the esophagus. This may account for the rarity of the reported cases. Myomas may be associated with narrowing of the esophagus and diverticula, but this is rare.

25. Guttman, M. R.: Primary Adenocystic Carcinoma or Cylindroma of the Trachea, Ann. Otol., Rhin. & Laryng. **45**:894-901 (Sept.) 1936.

26. Rose, J. D.: Myomata of the Oesophagus, Brit. J. Surg. **24**:297-308 (Oct.) 1936.

PETROSITIS

REPORT OF A CASE WITH A REVIEW OF SOME OF THE LITERATURE

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Today there is a great deal of discussion of, and much is written about, a rather recently recognized pathologic entity, especially from the point of view of diagnosis and treatment. A great deal of investigation has been done to clarify the understanding of petrositis, or infection of the petrous pyramid of the temporal bone, complicating otitis media with or without mastoiditis.

It would be presumptuous and also superfluous for me even to attempt to elaborate on this difficult and complex subject, since there are well qualified authorities who have had so much experience in every phase of the problem, including the macroscopic and microscopic anatomy, the pathology and the clinical picture, diagnosis and treatment.

However, I believe it is in order, in reporting another case, to refer briefly to some of the work that has been done, under the following headings: I. History; II. Anatomy; III. Pathology and Types; IV. Signs, Symptoms and Diagnosis; V. Roentgenograms; VI. Treatment, and VII. Complications.

I. HISTORY

Eagleton,[1] in 1929, was the first to operate on a patient with diagnosed suppuration of the petrous tip. He termed his method of operation the "unlocking of the petrous pyramid."[2] He also suggested the possibility of the translabyrinthine route.[2] However, operations had previously been performed to reach deep-seated abscesses originating in the petrous pyramid.

From the Department of Otology, Jefferson Hospital.

Read at the meeting of the Philadelphia Laryngological Society, April 5, 1938, College of Physicians, Philadelphia.

1. Eagleton, W. P.: Petrous Pyramid Suppuration: Fundamentals of Therapy, Ann. Otol., Rhin. & Laryng. **44**:1113, 1935.

2. Eagleton, W. P.: Unlocking of the Petrous Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex: Report of Four Cases with Recovery in Three, Arch. Otolaryng. **13**:386 (March) 1931.

As early as 1893, Bircher,[3] following a fistulous tract accidentally found, drained an abscess in the petrous pyramid, bringing about the recovery of the patient.

Streit,[4] in 1902, described the evacuation of pus in the region of the gasserian ganglion by an extrapetrosal approach after a radical mastoidectomy.

Goris,[5] in 1903, operating for a cerebral complication of chronic suppurative otitis media after performing a radical mastoidectomy, trephined in the temporal region; then, failing to find pus in the middle fossa, he removed more of the temporal bone, and by elevating the brain he found and removed a sequestrum.

In 1914, Broeckaert,[6] operating in a case of Gradenigo's syndrome, used an osteoplastic flap above the zygoma to reach the petrous tip.

In 1927, Richards[7] described an operation for approach to the petrous tip, but his approach was preceded by destruction of the labyrinth.

Mayer,[8] in 1929, operating in a case of acute purulent otitis media followed by Gradenigo's syndrome, used the extrapetrosal approach over the anterosuperior surface of the petrous pyramid after a radical mastoidectomy but failed to locate pus. After signs of meningitis appeared, pus was found coming from the floor of the tympanic cavity. With a large exposure here he discovered an opening about the orifice of the eustachian tube, filled with granulations. By probing into the petrous pyramid at this point for some distance a great deal of pus was evacuated, and the patient recovered.

Clarence Smith,[9] at the January 1931 meeting of the Section on Otology of the New York Academy of Medicine, reported drainage and cure of a subdural abscess of the petrous pyramid, having employed the anterior extrapetrosal route.

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3. Bircher, H.: Phlebitis des Sinus transversus, petrosus inferior und cavernosus: Eröffnung und Desinfektion nach Entfernung der Felsenbeinpyramide; Heilung, Centralbl. f. Chir. **20**:483, 1893; abstracted, Tr. Internat. Otol. Cong. **9**:314, 1912.

4. Streit, H.: Ein Operationsverfahren zur Eröffnung tiefgelegener, von der Spitze der Felsenbeinpyramide ausgehender Epiduralabscesse, Arch. f. Ohrenh. **57**:169, 1902-1903.

5. Goris: Un cas de cérébrale pour complication d'otite moyenne chronique, Ann. d. mal. de l'oreille, du larynx **29**:64, 1903.

6. Broeckaert, J.: Intervention par la voie supra-auriculaire en cas de paralysie otique du moteur oculaire externe associée a la névralgie du trijumeau, Rev. de laryng. **2**:209, 1914-1915.

7. Richards, J. D.: The Petrous Pyramid, Am. J. Surg. **2**:11, 1927.

8. Mayer, O.: Gradenigoscher Symptomkomplex bei akuter Mittelohreiterung: Operative Freilegung und Eröffnung eines Abscesses an der Pyramidenspitze in der Nähe des Bulbus venae jugularis; Heilung, Monatschr. f. Ohrenh. **63**:970, 1929.

9. Smith, C., cited by Kopetzky and Almour,[10] p. 399.

In 1930, Kopetzky and Almour [10] were the first to enter the petrous pyramid without opening the middle fossa, the point of entry being between the cochlea, the eustachian tube and the internal carotid artery. They entered with a burr "at an angle of between 20 and 25 degrees to the axis of the external auditory canal."

In 1933, Ramadier [11] described surgical procedures for drainage of suppurative apicitis in pneumatic bone.

In 1936, Eagleton [12] suggested the possibility of curing osteomyelitis of the inferior aspect of the petrous pyramid by entering the lateral cervical region and continuing through the retropharyngeal space. In 1937, Myerson, Blumberg and Rubin [13] described such an operation through the retropharyngeal space.

In 1936 [14] and 1937,[15] Lempert described a new technic for thorough exenteration of the apical carotid portion of the petrous pyramid, by exposure, through the anterior portion of the middle ear, of the internal carotid artery throughout its entire course in the bony canal of the temporal bone. By his procedure he can approach any part of the petrous tip it may be necessary to explore.

Since then several variations of the extrapetrosal approach have been described.[16]

Eves [17] stated: "Previous to 1929, very few cases were diagnosed before autopsy."

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10. Kopetzky, S. J., and Almour, R.: The Suppuration of the Petrous Pyramid: Pathology, Symptomatology, and Surgical Treatment, Ann. Otol., Rhin. & Laryng. **39**:996, 1930; **40**:157, 396 and 922, 1931.

11. Ramadier: L'ostéite profonde du Pocher, Oto-rhino-laryng. internat. **17**: 816, 1933.

12. Eagleton, W. P.: Osteomyelitis of the Inferior Surface of the Petrous Pyramid, J. A. M. A. **107**:482 (Aug. 15) 1936.

13. Myerson, M. C.; Blumberg, R., and Rubin, H. W.: A Proposed Operation for Osteomyelitis of the Inferior Aspect of the Petrous Pyramid, Arch. Otolaryng. **25**:381 (April) 1937.

14. Lempert, J.: Complete Apicectomy: Preliminary Report of a New Technic, New York State J. Med. **36**:1210, 1936.

15. Lempert, J.: Complete Apicectomy (Mastoidotympano-Apicectomy), Arch. Otolaryng. **25**:144 (Feb.) 1937.

16. Moorhead, R. L.: Suppuration of Petrous Pyramid: When and How to Operate; Report of Thirty Cases, Arch. Otolaryng. **28**:497 (Oct.) 1939. Jones, M. F.: Modification of the Semilunar Ganglion Approach Used in Surgery of Petrous Pyramid, read before the Section on Otolaryngology of the College of Physicians of Philadelphia and the Section on Otolaryngology of the New York Academy of Medicine, March 16, 1938.

17. Eves, C. C., cited by Mullin, W. V.: Certain Fundamentals in Regard to Suppuration of the Petrosal Pyramid: Fundamentals of Therapy, Ann. Otol., Rhin. & Laryng. **44**:1097, 1935.

II. ANATOMY

Myerson, Rubin and Gilbert [18] concluded, from the study of 200 specimens of the petrous portion of the temporal bone, that the perilabyrinthine space is not necessarily of the same anatomic structure as that of the mastoid of the same specimen.

Kopetzky [19] stated that at birth the perilabyrinth is diploic bone, with red or fatty marrow, and either pneumatizes or becomes sclerotic.

Guild [20] made the statement that the petrous pyramid is (*a*) "cancellous (spongy) bone," with red or fatty marrow or both, or (*b*) dense bone or (*c*) pneumatic cells or (*d*) a combination of all, and there are variations in size, shape and conformation. He stated also that the otic capsule is the only fixed and the "least variable" part.[20] Guild [20] and Tobeck [21] both stated that pneumatization develops from four points: (1) tubal, (2) hypotympanic, (3) epitympanic and (4) antral.

According to Guild the anatomic relations are as follows: above and behind, the superior petrosal sinus; below and in front, the carotid artery, jugular bulb and inferior petrosal sinus. Small vessels arising in the petrous pyramid empty into these various channels. "Nothing is known about the lymphatics of this region." [20]

Guild [20] stated also that, as is well known, the petrous apex "articulates with the body of the occipital bone and with the posterior angle of the greater wing of the sphenoid bone." He found occasional specimens showing *marked pneumatization* of these regions.

The relation between the location of pathologic processes and the involvement of the fifth to the eleventh nerve, the greater superficial petrosal and other nerves and the various ganglions and plexuses, producing characteristic clinical manifestations, was described by Guild.[20] He cautioned that "thorough knowledge of the variations in the detailed anatomy of the region is essential from the standpoints of pathology, of clinical diagnosis and of surgery."

The development of nasopharyngeal abscess in some cases was accounted for by Guild [20] by the fact that "Inferiorly the petrous pyramid . . . is very near to the lateral part of the vault of the nasopharynx."

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18. Myerson, M. C.; Rubin, H. W., and Gilbert, J. G.: Report on the Anatomical Study of Two Hundred Petrous Portions of the Temporal Bone, Laryngoscope **45**:159, 1935.

19. Kopetzky, S. J.: Acute and Chronic Otitis Media, Sinus Thrombosis and Suppuration of the Petrous Pyramid, Arch. Otolaryng. **24**:505 (Oct.) 1936.

20. Guild, S. R.: Anatomy, Normal and Pathological of the Petrous Pyramid, Ann. Otol., Rhin. & Laryng. **44**:1011, 1935.

21. Tobeck, A.: Anatomische Untersuchungen über die Pneumatisation von Felsenbein und die Wegleitung zur Spitze, Ztschr. f. Hals-, Nasen- u. Ohrenh. **37**:152, 1935; Ueber Pyramidenzellen und Pyramidenzelleneiterungen, Zentralbl. f. Ohrenh. **40**:193, 1935.

III. PATHOLOGY AND TYPES

Marvin Jones [22] showed by microscopic study of specimens the existence of osteomyelitis and osteitis and also empyema and that resolution takes place with fibrosis.

Fowler [23] made roentgen studies of the petrous bars, classifying and comparing them with microscopic sections likewise showing osteomyelitis, osteitis, empyema or a combination of all three.

According to Kopetzky [24] there are two forms of suppuration in the petrous pyramid: osteomyelitis (two types classified by Ramadier—hematogenic and otogenic) and coalescent osteitis.

According to Kopetzky,[25] "the invading organism has no etiological relationship to the lesion in question," the lesion in the petrous tip depending "upon the type of anatomical structure rather than upon the type of bacterial invader."

Seydell [26] used the same pathologic classification as Eagleton: [1]

1. Reactive and reparative osteitis.
2. Non-suppurative congestive cases—symptoms due to venous stasis.
3. Chronic bone sepsis cases (without macroscopic pus).
4. Abscess of apex.
   a. Without a tract.
   b. With a tract. (Acute or chronic—Seydell.)
5. Acute septicemia cases, associated with a continuous positive blood culture and meningitis.

The clinical classification as to location, made by Kopetzky [27] and Almour,[28] is most valuable, namely (as quoted from Almour):

1. Postero-superior petrositis.
2. Postero-inferior petrositis.
3. Postero-internal petrositis.
4. Anterior supracochlear petrositis.
5. Anterior subcochlear petrositis.
6. Anterior precochlear (pericarotid) petrositis.
7. Apicitis (empyema of petrous apex).

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22. Jones, M. F.: Normal and Pathological Anatomy of the Petrous Pyramid, Ann. Otol., Rhin. & Laryng. **44**:1036, 1935.

23. Fowler, E. P.: Normal and Pathological Anatomy of the Petrous Pyramid, Ann. Otol., Rhin. & Laryng. **44**:1056, 1935.

24. Kopetzky, S. J.: Diagnosis and Differential Diagnostic Data on the Specific Types of Suppuration in the Petrosal Pyramid, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1935, p. 89; Arch. Otolaryng. **22**:403 (Oct.) 1935.

25. Kopetzky, S. J.: Symptomatology and Diagnosis of Suppurations of the Petrous Pyramid, Laryngoscope **41**:398 (June) 1931.

26. Seydell, E. M.: The Clinical Picture of the Various Types of Infection in the Petrous Pyramid, Ann. Otol., Rhin. & Laryng. **44**:1068, 1935.

27. Kopetzky, S. J.: Petrous Pyramid Suppuration: Introductory Remarks, Ann. Otol., Rhin. & Laryng. **44**:1003, 1935.

28. Almour, R.: Petrosal Pyramid Suppuration: Fundamentals of Therapy, Ann. Otol., Rhin. & Laryng. **44**:1107, 1935.

IV. SIGNS, SYMPTOMS AND DIAGNOSIS

Kopetzky [24] stated that according to Ramadier there are two forms of *osteomyelitic lesion* in the petrous pyramid:

(*a*) The form called "hematogenic" is found most frequently in young patients. Clinically the patient presents the picture of general infection, marked prostration and gastroenteritic symptoms being present. Although otitic symptoms are present, they are overshadowed by other symptoms. Many times signs of pulmonary involvement hide the advance of the infection which is infiltrating the diploic bone and contained bone marrow of the petrous pyramid. It is almost inevitable that the lesion will reach the endocranial structures, the patient presenting, eventually, the symptoms of meningitis. The hematogenic form of osteomyelitic petrositis is usually bilateral.

(*b*) The second form is called "otogenic." This form of osteomyelitis is not confined to very young patients. Usually it occurs during the course of suppuration of the middle ear and is more easily shown to be a sequela of otitic infection. Commonly the onset occurs when the suppuration of the middle ear has become subacute. Its beginning is characterized by severe auricular and hemicranial pains on the same side of the head as the infected middle ear. The discharge from the middle ear itself becomes more profuse.

> The temperature ranges from 100 to 102 F. Sleep is disturbed, and often edema appears over the area of the upper attachment of the auricle. The walls of the external canal become edematous and painful to the touch. This tenderness of the periauricular region is in marked contrast to the absence of the usual signs of tenderness of the mastoid process. The painful edematous swelling spreads to the surrounding region, . . . [involving eventually] the upper cervical, the occipital and the temporomaxillary region.

Coalescent osteitis, according to Kopetzky,[24] "occurs only in pneumatized bone."

> [It] is a complication of infection of the middle ear. . . . Often the margins of these tracts of purulent cells break their way outward toward the tympanum and epitympanum and into the cavity of the mastoid wound, and are found as one or more fistulas. Since the development of this lesion is marked by the dominating signs and symptoms of coalescent mastoiditis, mastoidectomy has generally been performed long before the characteristic signs and symptoms of petrosal suppuration supervene.

Kopetzky [24] cautioned that one must differentiate this condition from other lesions. "Pain in the eye is sometimes noted in cases of thrombosis of a sinus, meningitis of the anterior fossa, and abscess of the brain, particularly in the temporosphenoid lobe, and . . . of involvement of the sphenoid and frontal sinuses."

Friesner and Druss [29] concluded from a histologic study in 24 cases of fatal meningitis:

> It might be timely, however, to reiterate that we have seen headache, pain in eye, fever and persistent otorrhea from an undrained zygomatic abscess; fifth and sixth nerve involvement from an epidural abscess over the mastoid tegmen; meningeal symptoms from a perisinus and epidural abscess or sinus thrombosis. These are some of the findings in the mastoid that we would consider adequate to explain pain. Of course a badly and extensively diseased mastoid is another.

Kopetzky [25] stated that the symptoms of suppuration of the petrous tip can be divided into four periods:

1. Period of pain in the eye (mostly at night) and aural discharge.
2. Period of low grade sepsis. (There may be vomiting and facial palsy.)
3. Period of quiescence. (Only fever occurs during this period.)
4. Terminal stage.

According to Kopetzky,[25] red blood cells and hemoglobin are not affected to any extent. The spinal fluid and eyegrounds are usually normal, and only moderate leukocytosis is present prior to the terminal stage.

Kopetzky [19] stated that "the Gradenigo syndrome is of peripetrosal origin, appears late in the course of the disease and is a meningeal reaction."

Vail [30] stated that the pain in the eye is caused by irritation of the great superficial petrosal nerve, rather than of the ophthalmic branch of the fifth.

The statements Kopetzky,[25] Lillie,[31] Seydell [26] and Eves [32] were all practically in agreement concerning the following symptoms:

1. Pain in and about the eye—especially at night.
2. Continuous aural discharge during the period of pain.
3. Low grade sepsis.
4. Onset of suppurative apicitis, usually late—an average of one month after a primary operation on the mastoid.
5. Palsy of the sixth nerve (not a definite part of the picture).

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29. Friesner, I., and Druss, J. G.: Petrosal Pyramid Suppuration: Fundamentals of Therapy, Ann. Otol., Rhin. & Laryng. **44**:1100, 1935.

30. Vail, H. H.: Vidian Neuralgia, with Special Reference to the Eye and Orbital Pain in Suppuration of the Petrous Pyramid, Arch. Otolaryng. **17**:212 (Feb.) 1933.

31. Lillie, H. I.: Petrosal Pyramid Suppuration: Summation and Conclusions of Symposium, Ann. Otol., Rhin. & Laryng. **44**:1129, 1935.

32. Eves, C. C.: Types of Petrosal Pyramid Suppuration, Ann. Otol., Rhin. & Laryng. **44**:1084, 1935.

6. Progress governed by changes in the frequency and character of pain, plus changes in the discharge.

7. Increasing involvement of petrous pyramid by infection as shown by a series of roentgenograms.

V. ROENTGENOGRAMS

Kopetzky [25] stated that of laboratory data obtainable the roentgenograms were the only real aid in the diagnosis of petrositis. This, of course, as Law [33] has mentioned, does not mean that diagnosis can rest on roentgen examination alone. Its limitations are attributed by Fowler [23] to the variable anatomy and the inaccessibility due to the depth of the petrous pyramid in the skull, as well as to the exceeding difficulty of interpreting the films.

Fowler [23] has said that roentgen evidence of pathologic change does not necessarily mean operation and vice versa.

According to Taylor [34] not every change in the petrous apex shown roentgenologically indicates a suppurative lesion of the petrous pyramid.

However, recognizing the limitations of roentgenographic aid to diagnosis, Nash [35] stated that

> X-ray studies are necessary in determining the anatomic structure of the petrosa, in estimating the amount and character of the destruction, and in ascertaining whether or not the bony confines of the petrosa are intact.

Lillie [31] said that it has been frequently suggested that the routine roentgenograms of mastoids should include the petrous pyramid.

Fowler [23] stated: "The roentgenogram may be of aid in the diagnosis of labyrinthitis or of labyrinthine irritation. At times definite evidence of pathology in the perilabyrinthine cells can be seen by x-ray."

Kopetzky [36] stated that

> Roentgenograms are the only means by which we can determine the advancement of decalcification. The finding of halisteresis is important. Secondary and subsequent x-rays determine its progress and add diagnostic data.

VI. TREATMENT

It is not my purpose in this paper to describe the technics of the various operations. Eagleton [1] concluded from his studies on the embryologic and anatomic structure of the apex that "infection within the

33. Law, cited by Lillie,[31] p. 1132.

34. Taylor, H. K.: The Roentgen Findings in Suppuration of the Petrous Apex, Ann. Otol., Rhin. & Laryng. **40**:367, 1931.

35. Nash, C. S.: Types of Petrosal Pyramid Suppuration, Ann. Otol., Rhin. & Laryng. **44**:1090, 1935.

36. Kopetzky, S. J.: Suppuration of the Petrosal Pyramid: Discussion, Ann. Otol., Rhin. & Laryng. **44**:1140, 1935.

petrous apex has a tendency to spontaneous subsidence, efficient drainage of the associated mastoid suppuration being all that is necessary to effect a cure."

Friesner and Druss [29] stated their agreement and also that "no one procedure is adequate to drain all foci in the pyramid."

Page [37] and Coates [38] agreed in stating (1) that not every acute mastoid infection with palsy of the sixth nerve should be treated radically and (2) that in only a small percentage of cases of diagnosed petrositis is radical surgical intervention necessary.

Kopetzky [39] stated: "Petrosal lesions are vastly different from each other, and no routine procedure is commonly applicable to all of them."

Lillie [31] said:

> One may well turn back to the dogma of Küster, who recommended that one should be prepared "to open up the bone freely so that it can readily be scrutinized, to clear away all disease and so fully to expose the source of suppuration that the pus is nowhere checked in its outflow." . . . one should approach this surgical problem prepared to attack the pyramid in an orderly manner without preconceived ideas or prejudices. One must not speak of conservative or radical surgical interference but should speak of the surgical interference as adequate and rational. . . . The optimal time for surgical interference may be difficult to determine. Each patient will always be a problem for individualized surgical judgment.

Page [37] stated:

> Interference with the lining membrane of the mastoid antrum and removal of the cells around the semicircular canals was discountenanced by some otologists not so long ago. . . . Since then it has become more generally the practice to look for softened structure in this region and to follow it to its depth, which explains the fact that men whose technic is thorough in this respect tell me that they rarely see complications at the petrous apex.

Almour [28] said that "differences of opinion between the various writers" are confined mainly "to the therapy indicated for the cure of the lesion." Each view "is true to a certain extent." Nomenclature "is the key to the fundamentals of therapy in suppurations of the petrous perilabyrinth." He described the various methods of approach, stating the advantages and disadvantages of each for the variously located lesions and pointing out that the type of operation depends on the location of the lesion.

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37. Page, J. R.: Suppuration of the Petrosal Pyramid: The Fundamentals of Therapy, Ann. Otol., Rhin. & Laryng. **44**:1093, 1935.

38. Coates, G. M.: Suppuration of the Petrosal Pyramid: Discussion of Symposium, Ann. Otol., Rhin. & Laryng. **44**:1142, 1935.

39. Kopetzky, S. J.: The Surgical Treatment of Various Types of Lesions in the Petrosal Pyramid, Surg., Gynec. & Obst. **66**:395, 1938.

VII. COMPLICATIONS

Seydell [26] listed complications as follows:

1. Meningitis. (Eagleton [40] said that frequently petrositis is the unrecognized cause.)

2. Facial paralysis.

3. Deafness (unusual) by involvement of the internal auditory meatus or labyrinth.

4. Abscess of the brain. (The literature says it is rare, but Seydell [26] recorded over 6 per cent.)

5. Impaired hearing. (The consensus is that there is no more than after operations on the mastoid.)

Eves [32] stated that 29 per cent of the patients died from complications in a series of 116 cases studied.

REPORT OF CASE

The patient, M. L., a Negro boy 9 years old, was admitted to Jefferson Hospital on Aug. 17, 1937, with a swelling and tenderness over the upper half of the right mastoid but none over the tip and a temperature of 100 F. There was a slight fulness of the posterior superior wall of the bony canal. The ear drum was slightly inflamed, especially the upper portion, and a little thickened, the picture being of the type one often sees in cases of receding otitis media. There was no discharge or perforation.

There was a history of intermittent earache on the right side for about three weeks. Swelling and tenderness over the mastoid developed on August 15. The left ear drum was normal. No history of previous aural trouble was elicited, and the patient had always been in good health. He was well nourished. The remainder of the examination gave negative results.

The blood count on admission was 12,450 leukocytes, 70 per cent hemoglobin, 3,550,000 erythrocytes, color index 1.

Roentgenograms of the mastoid showed the cellular structure on the left well maintained, but the appearance of the cells on the right indefinite on account of motion. Roentgenograms showed the heart and lungs normal.

On August 18, a mastoidectomy was done, revealing a mixed type of cellular structure, little free pus, some breaking down of the walls of the cells and marked softening in the areas of the zygoma and the posterior angle and even over part of the cerebellum, necessitating counterincision and exposure of the dura over the cerebellum. Disease of the tegmen required exposure of the middle fossa and a large portion of the sigmoid sinus, revealing a normal dura and slight discoloration of the sinus at its lower end.

The organism was Streptococcus haemolyticus.

After operation the temperature was 100 F.

For five days the temperature was a low grade septic type. The fifth postoperative day it was between 99 and 101 F. The packing was removed for the first time and clips removed. A fair amount of discharge was present. On the ninth postoperative day, on close questioning, the patient admitted having headache and pain in the left frontal region and about the left eye. Some discharge continued;

40. Eagleton, W. P., cited by Seydell,[26] p. 1070.

the temperature remained slightly elevated, and the general condition seemed good, but on August 31 he still stated, on questioning, that he had pain in the left eye and headache in the left frontal region. There was no record of pain being worse at night. He was certainly not irritable.

On September 1, or the fourteenth postoperative day, the temperature rose to 104.8 F. and for twelve hours remained 104 F. In three hours, on September 2, it dropped to 99 F. and in six more was up to 103 F. The patient then displayed paralysis of the right sixth nerve, with pain in the eye on the opposite side and slight nuchal rigidity, a slight bilateral Kernig sign and a suggestion of ankle clonus bilaterally. A spinal tap yielded a turbid fluid, reducing copper and containing 19 mg. of sugar, a marked increase of globulin, 1,400 cells, no organisms, 26 per cent lymphocytes and 74 per cent neutrophils; the Wassermann reaction was negative. Culture showed no growth in forty-eight hours. By the next day, September 3, the temperature had dropped to 100 F., when another operation was performed.

Roentgenograms taken on September 2 showed clouding of the cellular structure of the right petrous pyramid compared with that of the left, with intact surfaces and no destruction of bone.

On September 3, a radical mastoidectomy was performed and a search made for a tract to the petrous pyramid. The former wound had healed nicely, except for a small opening at the lower end for drainage. The sinus and exposed dura were covered with healthy, organizing granulations. The posterior incision was extended further. A goodly portion of the squamous bone and all the inner table of the mastoid were removed, a large exposure of the middle and posterior fossae and a larger exposure of the cerebellum resulting. There was some osteomyelitis of the occipital bone; this was therefore removed until good bone was reached. The entire sinus was uncovered from beyond the knee to the jugular bulb. Peribulbar cells, cells internal to the styloid process and the entire tip were removed. Although there was disease in these areas, no fistulous tract was found. The semicircular canals were skeletonized, the perilabyrinthine structure being removed as far as possible. The dura was further exposed from the zygomatic area down to the middle ear. The promontory appeared intact, and there were no fistulas in the labyrinth. The mouth of the eustachian tube appeared large in caliber. Since there appeared some necrosis about the tube, peritubal cells were curetted and found diseased, a finding which necessitated the opening of a large area around the ascending portion of the carotid artery. However, no free pus appeared. Since there appeared to be no further tract leading inward, it seemed wise to stop there. There seemed to be no fistulous tract in the posterosuperior, the posteroinferior, the posterointernal, the anterior supracochlear or the anterior subcochlear region. The anterior precochlear, or peritubal, or pericarotid, region was the only suspicious point.

The brain was then elevated from the anterosuperior surface of the petrous bar and a probe passed along the bar to the tip; as nothing abnormal was felt and no pus evacuated, it was felt that it was unnecessary to investigate further and that all necessary points had been inspected and thorough drainage established. Under the conviction that suppurative apicitis was not present, the operation was stopped there and the wound left wide open and drained with iodoform gauze, the plastic work of making flaps being left for a later date, if the patient survived.

The next day the temperature went as high as 102 F., and it stayed around 101 F. for three days. The pain and headache subsided, but the palsy of the sixth nerve remained.

The patient's recovery was uneventful. From September 6 to October 22, when the third operation was done to complete the plastic work, the temperature, pulse and respiration remained more or less normal.

On September 21, a spinal tap was done merely for a check-up, and the findings were normal throughout, with 14 cells per cubic millimeter.

Roentgenograms taken on September 22 showed that the cellular structure of the right petrous pyramid was still cloudy compared with that of the left.

It was unfortunate for the records that the second operation was undertaken without waiting for examinations of the eye, but of course consideration for the patient was the first thought. On September 20, a check-up made in consultation by the eye department showed loss of function of the right external rectus muscle, with the remaining extraocular muscles functioning normally and the eyegrounds normal.

By November 1, the palsy of the sixth nerve was entirely cleared up. The patient was kept in the hospital another month, because he lived in Delaware. He was discharged on November 24, with the posterior wound entirely healed and all symptoms relieved. On Jan. 25, 1938, the family doctor wrote me that the boy had been perfectly well and had been in school since January 1.

The interesting feature of this case, to me, at least, was the fact that the surgical procedure was of a radical type from one point of view and yet constituted a so-called conservative operation from another. It was radical from the point of view of the amount of bony structure removed and yet conservative as contrasted with the so-called radical procedure for petrositis. As much as possible of the temporal bone up to the basal labyrinthine portion of the petrous pyramid was removed. The question arises: Was this extensive surgical intervention necessary or not? I believe the radical mastoid procedure was certainly indicated, to investigate the pericochlear and peritubal area, and justified by the evidence of pathologic change in the peritubal area. Whether or not the rest of the procedure was necessary I cannot answer.

Another question arises: Was the pathologic condition causing the clinical picture in the peritubal, the peribulbar or the perilabyrinthine area?

COMMENT

A brief review of the principal features of petrositis, gathered from some of the literature, is presented.

Another case of petrositis is presented, in which the frontal headache and the pain in the eye occurred on the side opposite the lesion and the palsy of the sixth nerve.

Emphasis should be made on a point taken by Page,[37] that thorough surgical intervention about the antrum and the semicircular canals and the following to its depth of all softened structure will obviate many complications in the petrous apex.

Medical Arts Buidling.

ROLE OF THE TONSILS IN EXPERIMENTAL ENDOCARDITIS

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AND
MARGERY BLAHD, M.D.
CHICAGO

The advisability of removing the palatine tonsils as a prophylactic measure for the various forms of endocarditis has long been a subject of controversy among clinicians. The advocates of tonsillectomy point out the decreased incidence of chronic cardiac disease in tonsillectomized persons, as borne out by certain studies.[1] Their opponents, basing their opinions on studies of other groups, maintain that routine removal of tonsils for the prevention of cardiac disease is not based on conclusive data.[2] Furthermore, some clinicians contend that actual harm can be done by removal of the tonsils,[3] not only in the operative mortality but also in respect to the development of infections of the respiratory tract.

In a previously reported study,[4] it was shown that a virulent hemolytic streptococcus obtained from a pneumonic lesion in a dog produced endocarditis in 10 of 25 dogs, i. e., in 40 per cent when injected intravenously. In view of these relatively successful results, it was decided to introduce these same bacteria into the tonsils of dogs and determine whether endocarditis could thus be produced. To study further the role of the tonsil in the development of endocarditis, it was planned to inject these streptococci into the tonsillar beds of dogs which had previously undergone tonsillectomy.

The use of hemolytic streptococci was furthermore appropriate because of their frequent isolation from the tonsils of patients with endocarditis.[5]

From the Department of Pathology of the Nelson Morris Institute, Michael Reese Hospital.

1. Kaiser, A. D.: Influence of Tonsils on Rheumatic Infection in Children, J. Lab. & Clin. Med. **21**:609, 1936.

2. Wilson, M.; Lingg, C., and Croxford, B.: Statistical Studies Bearing on Problems in the Classification of Heart Disease, Am. Heart J. **4**:197, 1928.

3. A Criticism of Tonsillectomy, Foreign Letters (London), J. A. M. A. **111**:181 (July 9) 1938.

4. Blahd, M.; Frank, I., and Saphir, O.: Experimental Endocarditis in Dogs, Arch. Path. **27**:424 (March) 1939.

5. Davis, D. J.: Bacteriology and Pathology of the Tonsils with Especial Reference to Chronic Articular Renal and Cardiac Lesions, J. Infect. Dis. **10**: 148, 1912.

METHODS

The organism was a beta hemolytic streptococcus isolated from a pneumonic lesion in a dog. The bacteria were in the smooth phase on blood agar. They fermented lactose and salicin but not mannitol or sorbitol. There was a slight formation of acid in the presence of trehalose. The organism was a group C streptococcus according to the Lancefield precipitin test.[6]

The virulence of the streptococci was maintained by constant passage through the blood stream of dogs. Eighteen to twenty-two hour cultures of these bacteria in beef heart dextrose broth were used. Each cubic centimeter of broth contained an average of one and one-half to two billion bacteria.

The palatine tonsils of 30 dogs of the variety usually supplied to the laboratory were injected one to three times per week. Quantities of culture varying from 1 cc. to the sediment of 20 cc. were thoroughly shaken and injected with a 10 cc. syringe bilaterally into the tonsillar and peritonsillar tissues, with the animal under anesthesia induced by intravenous injection of pentobarbital sodium.

Those animals which did not die spontaneously were killed after a four to twelve week course of injections. All the dogs in our previous study in which endocarditis developed had succumbed in less than this period. Autopsies were performed as soon as possible after death, at which time cultures of the heart blood in dextrose broth were made. Microscopic sections were taken from the tonsils, heart and kidneys and stained with hematoxylin and eosin.

The control group consisted of 15 dogs, whose tonsils were removed with a wire loop, with the animals under anesthesia induced by intravenous injections of pentobarbital sodium. Injections were made into the tonsillar beds and the parapharyngeal tissues, but at least one week was allowed to elapse after tonsillectomy, to insure complete healing. These dogs were treated identically as the experimental series, except that the culture material was introduced into the parapharyngeal tissue rather than the tonsil.

RESULTS

Of the 30 dogs which received intratonsillar injections, outspoken endocarditis developed in 2.

One of these revealed acute thromboulcerative endocarditis of the aortic valve, with septic infarcts in the kidneys and spleen, acute myocarditis and bronchopneumonia. The tonsils were soft, and histologic examination disclosed acute inflammation, with polymorphonuclear infiltration of the peritonsillar tissues. This animal had received fourteen injections over a period of four weeks, each injection averaging 7 cc. of culture material divided about equally between the two tonsils.

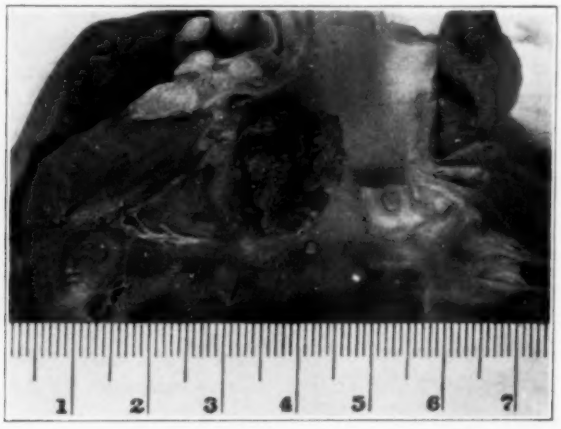
The other dog, during two and one-half weeks, received eight injections of from 2 to 4 cc. of culture material in each tonsil. Postmortem examination of this animal revealed verrucous endocarditis of the aortic leaflet of the mitral valve, acute myocarditis, acute focal glomerulonephritis and acute membranous tonsillitis. The culture of the heart blood of both dogs was positive for the hemolytic streptococcus.

6. Topley, W., and Wilson, G.: The Principles of Bacteriology and Immunity, ed. 2, Baltimore, William Wood & Company, 1937.

Autopsy of the other 28 dogs disclosed no pertinent findings other than bronchopneumonia and evidence of acute or chronic tonsillitis and peritonsillitis. In all animals, markedly swollen and inflamed tonsils (acute tonsillitis) had developed early in the course of the injections. In several, this condition subsided after a short time, despite repeated injections. After two to three weeks the tonsillar tissue appeared atrophic.

The heart blood of 5 dogs in which endocarditis did not develop contained hemolytic streptococci.

Endocarditis did not develop in any of the control group of 15 tonsillectomized dogs. All of them showed marked acute inflammation and swelling of the pharyngeal tissues, at least early in the course of injections. Postmortem examination revealed acute and chronic inflammation



Huge vegetations involving the right and a portion of the left aortic cusp and smaller vegetations on the posterior cusp.

and edema of the parapharyngeal tissues, and in 7 instances parapharyngeal abscesses were observed. Repeated cultures of the blood of these animals remained sterile.

COMMENT

The development of endocarditis in 2 of 30 dogs by intratonsillar inoculation of our strain of streptococci is significant in the light of the negative observations on the tonsillectomized dogs which had received injections of this same organism in the tonsillar bed. Although the number of positive experimental results appears small at first glance (2 of 30), it must be remembered that in patients also the incidence of endocarditis as compared with that of tonsillitis is small.

The possibility that the culture material may have been introduced directly into the circulating blood must be considered. At the time

of the tonsillar inoculations some of the bacteria may have been injected into the tonsillar vessels, with resulting bacteremia. This possibility is especially significant in view of the fact that in a previous study we were able to produce endocarditis in dogs even by a single intravenous injection of the same streptococcus.[4] If this possibility were a fact, we might expect some of the injected material in the control animals likewise to have reached the blood stream. However, neither bacteremia nor endocarditis was observed in any of the control series. Furthermore, after every injection of the culture material in either the tonsils or the peritonsillar regions, there was marked ballooning of the tissues, indicating that the broth accumulated locally and did not escape into the blood vessels. Apparently the tonsil in some way fostered the entrance of the streptococci into the blood stream. This may result from the concentration of large numbers of lymphatics or perhaps from a specific distribution of blood vessels, and it does not result if the tonsils have been removed.

It would be hazardous to draw definite conclusions from this relatively small series of animals or to apply these results too readily to human beings. However, it is obvious from these studies that bacteria from the tonsils are able to invade the circulating blood and to produce endocarditis. It is therefore possible that in the human being also inflammation of the tonsils plays a definite role in the development of endocarditis. The value of tonsillectomy in actually preventing endocarditis in the human being or at least in reducing the incidence of this disease cannot be accurately estimated from these experiments. However, the fact that no endocarditis was obtained in the animals whose tonsils were removed indicates the advisability of tonsillectomy in the prophylaxis of endocarditis.

SUMMARY AND CONCLUSION

In 2 of 30 dogs, after intratonsillar injection of virulent beta hemolytic streptococci acute endocarditis developed. The hemolytic streptococci used had previously produced endocarditis in 40 per cent of a series of 25 dogs.[4] Endocarditis did not develop in any of 15 tonsillectomized dogs after repeated introduction of the same organism into the tonsillar bed and parapharyngeal regions. Furthermore, repeated blood cultures of the control dogs remained sterile. It is likely that the tonsils played a role in the development of bacteremia and endocarditis in these experiments. The results of the investigations cannot be applied too readily to the patient. However, these studies present experimental confirmation of the favorable clinical results reported in the literature in respect to prophylactic tonsillectomy for endocarditis.

THE PETROUS APEX OF THE TEMPORAL BONE AND ITS RELATIONS

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Every one has so-called individual characteristics and definite susceptibilities to disease—susceptibilities which may be determined by endocrine or allergic reactions or by anatomic constitution and arrangements which make possible definite pathologic sequelae. In the respiratory, the cardiovascular, the gastrointestinal and the urinary system these susceptibilities are fairly well known, but the nose, the sinuses and the pneumatic temporal bone, although they may be considered adjuncts of the respiratory system, often present problems which are baffling and apparently not correlated with the patient's general constitution. Individual problems, then, arise in each case, and anatomic variations must be constantly kept in mind, even though they may not be extreme. Unusual anatomic arrangements which depart little from the so-called normal may place a nerve, an artery, a vein or a venous sinus in a vulnerable position. This abnormality may result in unusual or confusing symptoms, which may be interpreted accurately and correctly only if detailed and accurate anatomic information is available and the possibility of variations from the "normal" is taken into account.

For the past few years, the temporal bone has received much attention; there has been considerable discussion concerning the interpretation of the signs and symptoms arising from or accompanying an inflammatory process in this area, and much difference of opinion has arisen as to cause and effect.

The temporal bone is unique in its connection with adjoining bones. The sutures tend to remain open, with fibrous tissue filling the gaps. The upward-forward part of the squamous portion is beveled over a wide surface to articulate with a similar area on the parietal and outer

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Mr. A. B. Streedain prepared the illustrations, and the contributions of the Research Study Club of Los Angeles to the Anatomy Research Fund aided in this study.

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surface of the greater wing of the sphenoid. The lateral border of the greater wing, in front of the spinous process, also is beveled to receive the lower border of the squama. It is interesting to note also that the zygomatic process is beveled in such a way as to rest on top of the temporal process of the malar bone. The petrous apex is loosely attached to the posterior border of the greater wing and to the basilar process of the sphenoid and the occipital bone. By this arrangement one will observe that a certain amount of movement from below upward

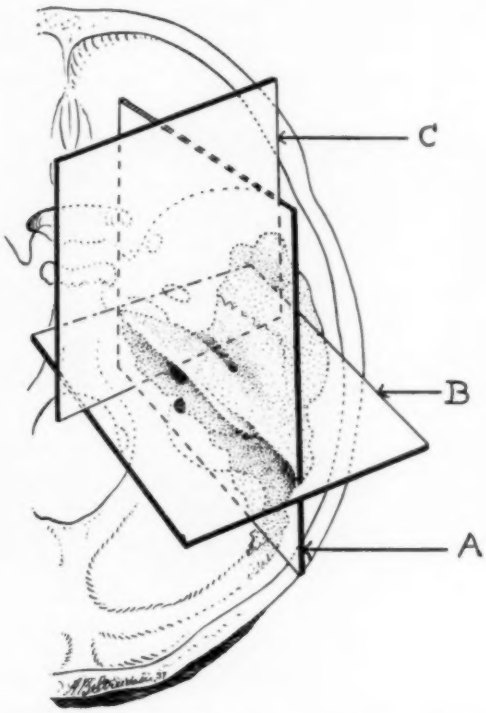

Fig. 1.—A diagrammatic representation of the right half of the base of the skull seen from above, to show the relative position of the temporal bone and the planes in which sections have been made. *A* is the vertical plane, almost parallel with the cerebellar or posterior surface; *B*, the horizontal plane, almost parallel with the cerebral or superior surface, and *C*, the transverse plane, which is at a right angle to the long axis of the pyramid.

is provided for, the hearing apparatus thus being protected against possible damage from forcible closure of the jaws, especially during the period of dentition, when occlusion is imperfect, or perhaps against shock which might result from a blow on the chin. The arrangement allows also for molding during birth with minimum traction on the vital parts of the organ of hearing. The labyrinthine capsule grows

little or none after birth, but the other portions of the petrous bone, especially the apex, develop rapidly to keep pace with the increase in width and length of the base of the skull. The open sutures and the beveled arrangement of the bone's edges make it possible for this growth to take place without undue stress on the otic capsule.

I wish to discuss the anatomy of that portion of the pyramid of the temporal bone which in the literature is designated the petrous apex—the carotid portion (fig. 2). It includes all the temporal bone mesial to the labyrinthine capsule and is in relation to the internal carotid artery. Within this portion is found the cellular structure which is involved in inflammation of the apex. I shall point out the variations in the structure of the apex itself, as well as the variations

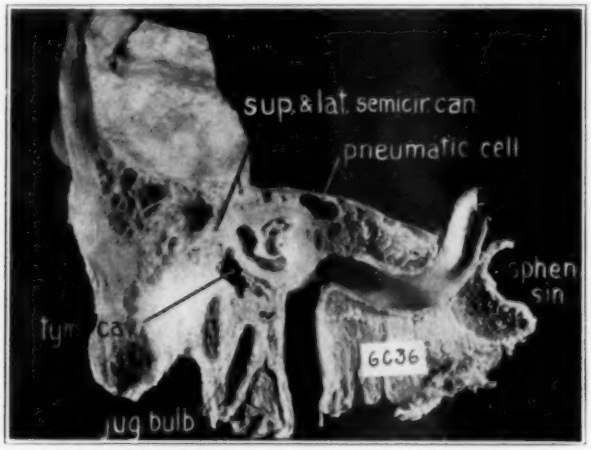

Fig. 2.—A vertical section through the left temporal bone, extending through the sphenoid, showing the course of the internal carotid artery. The cell tract under the superior semicircular canal extends from the mastoid antrum to the subarcuate fossa, where a large pneumatic cell is seen. This section shows the tympanic cavity and vestibule opened, as well as the basal coil of the cochlea, above which the auditory and facial nerves are seen. The section is cut in plane *A* (fig. 1).

in the arrangement of neighboring structures, an understanding of which helps to explain the multiplicity of clinical pictures.

The apex is placed between the greater wing of the sphenoid in front and the basilar process of the occipital bone behind and is directed forward and mesially (fig. 3). For purposes of description it may be said to have an anterior, a posterior and an inferior surface and an anterior, a posterior and a superior border; but for the present purpose it seems best to consider the surfaces from a clinical point of view and refer to them as cerebral, cerebellar and extracranial.

The floor of the middle cranial fossa is an inch (2.5 cm.) or more higher than the floor of the posterior fossa, and the pyramid of the temporal bone is so placed as to form the step-up, as it were, the cerebellar surface being the rise and the cerebral surface the tread of the step. The cerebral surface looks upward and forward and forms a part of the floor of the middle cranial fossa, on which rests the temporal lobe of the cerebrum. Along the forward portion there are two grooves running parallel with its long axis, one for the greater and one for the lesser superficial petrosal nerve. If the cortex of the bone should be removed at the beginning of these grooves the tympanum would be uncovered near the opening of the eustachian tube. The foramens for the petrosal nerves would then offer a direct line for the transmission of

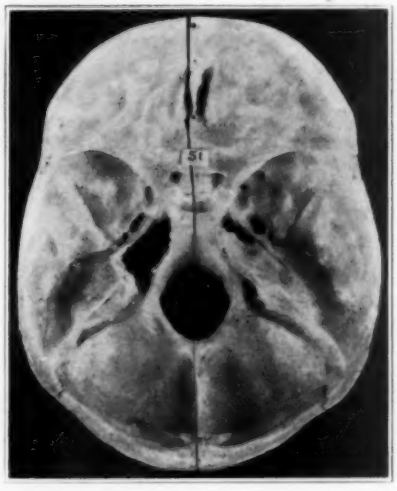

Fig. 3.—Base of the skull seen from above. The left petrous apex has been removed by a section extending from the hiatus Fallopii to the notch for the exit of the cochlear duct.

infection from the middle ear to the temporal fossa, in which event the greater superficial petrosal nerve might be involved and give rise to disturbance in lacrimal and nasal secretion. Through the vasomotor fibers of the vidian nerve there might be an alteration in the blood supply to the region of the lacrimal gland, along with similar disturbances over the posterior part of the nose and palate. Herpetic eruptions over this area might also follow. Some writers [1] have expressed the opinion that fibers for deep pain sense are included in this nerve and

1. Vail, H. H.: Vidian Neuralgia, with Special Reference to Eye and Orbital Pain in Suppuration of the Petrous Apex, Arch. Otolaryng. **17**:212 (Feb.) 1933.

that this fact accounts for the severe pain back of the eye which so often accompanies inflammation of the apex. The lesser superficial petrosal nerve, if affected, might produce a perverted secretion and altered blood supply to the parotid gland, with herpes in front of and below the ear. A branch of the middle meningeal artery enters the tympanic cavity by way of the aquaeductus Fallopii, which also may, by retrograde arteritis or periarteritis, allow infection from the middle ear to enter the temporal fossa. Often a considerable portion of this surface is wanting, being replaced by fibrocartilage over the internal carotid artery. The anterior border articulates with the greater wing of the sphenoid. Along it lies the eustachian tube, immediately behind which is the carotid canal, with only a thin lamella of bone separating the two. At the mesial end of this border there is an irregular opening, the foramen

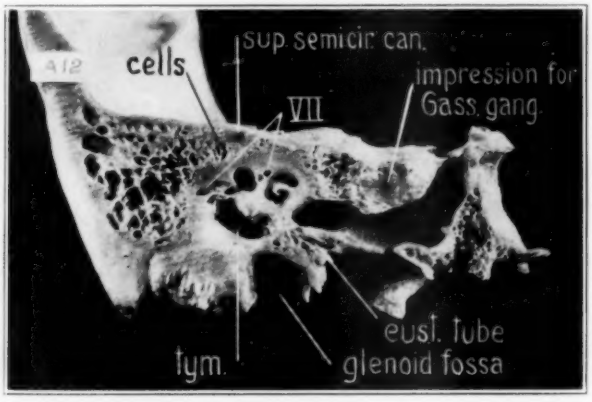

Fig. 4.—A vertical section through the oval window of the right temporal bone, viewed from the front, showing the extension of cells from the mastoid region into the petrous apex through the arch of the superior semicircular canal. The cortex in the floor of the gasserian impression is thin and perforated by many small foramens for the transmission of veins. The notch at the tip is for the abducens nerve as it passes through Dorello's canal. The basal coil of the cochlea is located at the bend in the carotid canal above which two openings are seen, for the facial and the auditory nerve. Below the lateral semicircular canal a portion of the facial canal is open. The posterior wall of the osseous eustachian tube is seen along the carotid canal. The section is cut in plane *A* (fig. 1).

lacerum, for the passage of the internal carotid artery with its sympathetic plexus and of the pericarotid venous plexus, connecting the cavernous sinus with the pterygoid plexus.

An important point for consideration in connection with this portion of the apex is the small fossa, or impression, for the gasserian ganglion (figs. 4, 5 and 6). Here the cortex of the bone is thin and often perforated by a number of small openings. In life these openings transmit veins, which drain the cellular structure within and connect the

Fig. 5.—A horizontal section of the right temporal bone, showing the cellular structure of the petrous apex. Note the thin bone between the cells and the gasserian ganglion, which has been lifted from its bed. The section is cut in plane *B* (fig. 1).

Fig. 6.—Drawing of a skull, showing extensive erosion of the left petrous apex at the impression for the gasserian ganglion, where the cortex is often thin and perforated for small veins. Destruction of bone is shown also in the region of the knee of the transverse sinus.

venous plexus surrounding the gasserian ganglion with the pericarotid plexus. Thus infection may be transmitted directly to the ganglion and give rise to severe deep-seated pain early in disease of the bone. On the other hand, if this area drains into the veins around the internal carotid artery (fig. 7) the carotid sympathetic plexus will first be irritated, and a different chain of symptoms will rise, viz., pupillary disturbance and alteration of the blood supply to the orbital structures, nose, nasopharynx, soft palate or pharynx. Occasionally the chief drainage is into the first part of the inferior petrosal sinus, which would expose the abducens nerve to early involvement, or it may be into the petro-occipital sinus, which would carry the infection toward the jugular

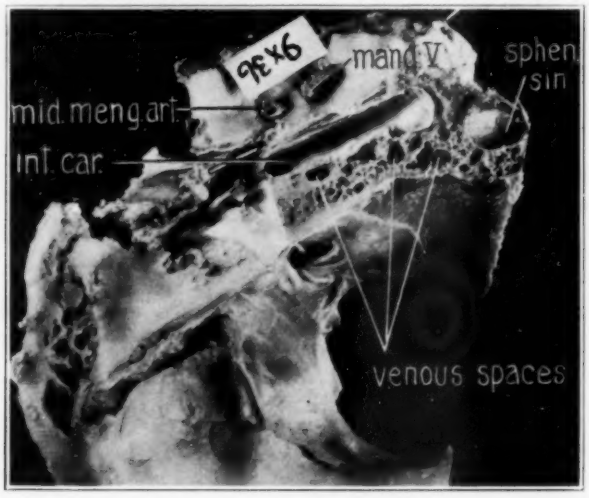

Fig. 7.—A horizontal section of the left petrous apex, showing the continuity of the cellular structure of the apex with that of the sphenoid. Some large cells along the internal carotid artery are venous spaces. The mandibular nerve and the middle meningeal artery are sectioned at their foramens. The probe is inserted in the eustachian tube. The section is cut in plane *B* (fig. 1).

bulb. As shown by Eagleton,[2] infection may be carried by the carotid venous plexus directly to the cavernous sinus.

The cerebellar surface of the apex is almost vertical and forms a part of the floor of the posterior cranial fossa. The opening of the internal auditory meatus marks its lateral boundary. Infection may be carried to the cerebellar fossa along the dural sheath surrounding the facial and the auditory nerve, as well as along the blood vessels which accompany them. Just lateral to and a little above the internal auditory

2. Eagleton, W. P.: The Carotid Venous Plexus as the Path of Infection in Thrombophlebitis of the Cavernous Sinus: Its Relation to Retropharyngeal and Sphenoidal Suppuration, Arch. Surg. **15**:275 (Aug.) 1927.

meatus is an irregular depression, the subarcuate fossa, which is usually perforated by one or two openings for the passage of an artery and vein, along which infection may travel (fig. 2).

The extracranial, or inferior, surface is irregular and is located outside the skull at the upper end of the carotid sheath. An abscess perforating the cortex of the bone at this point would extend along the great vessels of the neck or perhaps point to the lateral pharyngeal recess or form a retropharyngeal abscess.

Fig. 8.—Drawing of a skull, showing a wide petrobasilar suture allowing for free communication between the inferior petrosal and the petro-occipital sinus. In this case the petrous apex would be almost completely surrounded by venous blood.

The posterior border, or petrobasilar suture, is curved from above downward and outward. This marks the course of the inferior petrosal sinus on the inside, and the petro-occipital sinus, or sinus of Englich, on the outside, of the skull. Both channels connect the cavernous sinus with the upper end of the jugular vein. Along this border the apex comes into relation with the side of the body of the sphenoid for a

distance of 3 to 7 mm. and with the basilar process of the occipital bone for the rest of its length. This suture may be wide, with deep grooving above and below for the blood channels and only a thin fibrous partition between the two (fig. 8). Occasionally this suture is completely ossified, the diploe of the sphenoid and of the occipital bone being brought in direct continuity with that of the apex (figs. 9 and 10). Thus osteomyelitis may extend in either direction at this point.[3] Below the internal auditory meatus a notch marks the exit of the cochlear duct, which is accompanied by a small artery and vein, either of which may serve as a channel for the extension of infection. The petrous ganglion of the glossopharyngeal nerve is located in the fossa and may be affected and give rise to a disturbance in the sense of taste on the posterior third of the tongue.

The superior border or crest is the highest point of the petrosa and forms the boundary between the posterior and the middle cranial fossa. A groove marking the course of the superior petrosal sinus can usually be seen extending the full length of the crest. As it approaches the mesial end it curves downward to pass below the notch for the trigeminal nerve. This border gives attachment to the tentorium cerebelli, which is firmly adherent to the bone except at the mesial end, where it extends upward to be attached to the posterior clinoid process. One layer is reflected downward to become continuous with the dura of the posterior cranial fossa; the other extends forward, attaching itself to the anterior clinoid process, and is reflected downward, forming the lateral wall of the cavernous sinus and becoming continuous with the dura in the floor of the middle cranial fossa. In the angle formed by this arrangement the cavernous sinus extends backward to form the superior and the inferior petrosal sinus. The posterior bend of the internal carotid artery is located in this space, against the side of the body of the sphenoid under the posterior clinoid process (fig. 11). The trigeminal nerve, in its course forward over the crest, passes through this fold of dura, carrying with it a heavy dural sheath. In doing this, the nerve often divides the superior petrosal sinus into two channels, one above and one below the nerve; the two join lateral to the nerve, to continue along the crest of the bone as the superior petrosal sinus. The triangular space between the fifth nerve and the side of the quadrangular plate of the sphenoid is divided by the petrosphenoid ligament, or ligament of Gruber, into an upper and a lower compartment (fig. 12).

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3. Lawson, L. J.: Osteomyelitis of the Sphenoid Bone: Report of Two Cases, Arch. Otolaryng. **25**:1 (Jan.) 1937. Halle, M.: Review of a Case of Osteomyelitis of the Sphenoid Bone Extending to the Petrous Apex Through the Mastoid Process with Exenteration of the Middle Ear in 1900, Laryngoscope **48**:198, 1938. Richardson, C. W.: Osteo-myelitis of the Temporal Bone, Tr. Am. Otol. Soc. (pt. 1) **12**:126, 1910.

Fig. 9.—Drawing of a skull, showing the right petrous apex fused with the basilar process. The deep impression for the trigeminal nerve and the gasserian ganglion is shown.

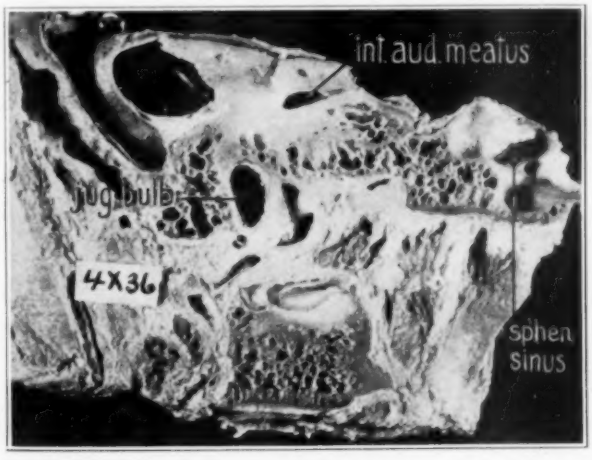

Fig. 10.—A vertical section of the temporal bone, showing the continuity of the diploe of the petrous apex with that of the sphenoid across the petrobasilar suture. The section is cut in plane *A* (fig. 1).

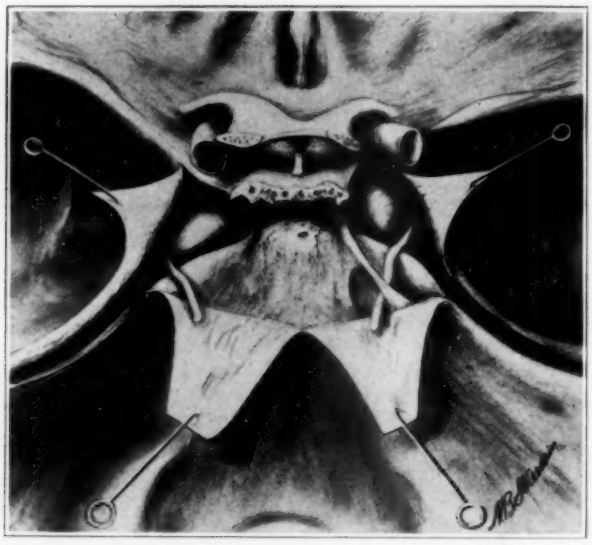

Fig. 11.—Drawing of a dissection, showing the dura incised and retracted and the posterior part of the cavernous sinus exposed, as well as the posterior bend of the internal carotid artery. The abducens nerve bends at a right angle over the high, sharp upper border of the petrous apex. The petrosphenoid ligament is absent on the left side and represented on the right by a small, strong band, which makes an indentation in the nerve where it is compressed against the bone. Blood going to the inferior petrosal sinus passed entirely above the ligament.

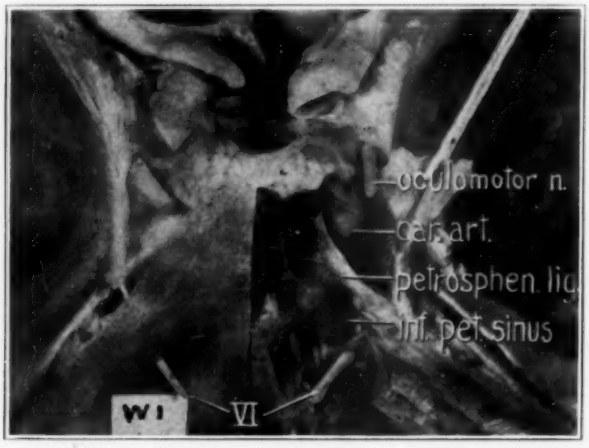

Fig. 12.—Photograph of a dissection, showing Dorello's canal. The abducens nerve is seen as it crosses the inferior petrosal sinus to run along its lateral wall. The petrosphenoid ligament is a well developed structure in this specimen. The section shows the trabeculae for the basilar plexus, which serves as a communication between the petrosal sinuses of the two sides.

The ligament extends from the accessory posterior clinoid process to some point on the pyramid. There is often a small spine on the upper border near the tip, which marks its attachment (fig. 13). This ligament varies in length, size and constancy. It may be present on one side and absent on the other. The notch below this ligament is known as Dorello's canal [4] and contains the inferior petrosal sinus and the abducens nerve. The blood destined to pass into the inferior petrosal sinus may all pass below the ligament (through Dorello's canal), may pass above or may leave by both routes, joining below the ligament to form the inferior petrosal sinus, at which point there is usually free communication, through the basilar plexus, with its fellow on the oppo-

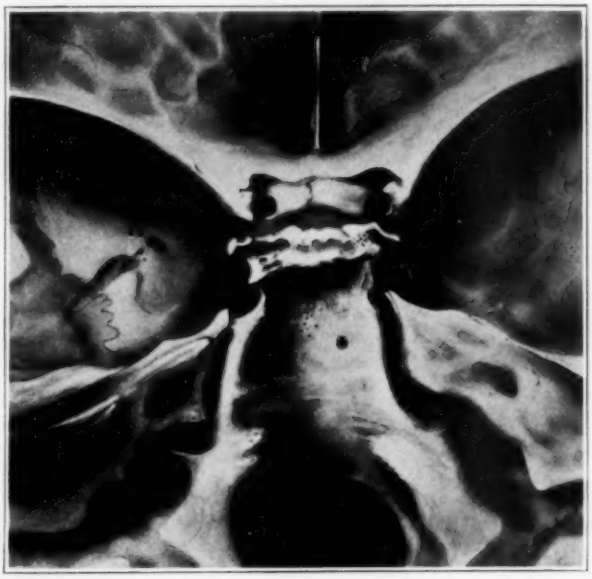

Fig. 13.—Drawing of a skull to show the relation of the petrous apex to the basilar process. The sphenoid spine on the tip of the pyramid and the accessory posterior clinoid process are plainly seen. To these the petrosphenoid ligament is attached to complete Dorello's canal. This notch is wider on the left than on the right. The deep groove for the inferior petrosal sinus and the wide petrobasilar suture are shown. The left jugular foramen is divided by a bony septum into an anterior and a posterior compartment.

site side, which might account for involvement of the abducens nerve on the contralateral side. The size of this sinus varies from a narrow channel to one 7 mm. in width. The abducens nerve pierces the dura some distance below the crest on the mesial side of the inferior petrosal

4. Vail, H. H.: Anatomical Studies of Dorello's Canal, Laryngoscope **32**: 569, 1922.

sinus and usually crosses the sinus to continue upward through Dorello's canal on the lateral side, where an impression in the apex is often seen. This relation is not constant, varying with the width of the dorsum sellae and the basilar process of the occipital bone. If the basilar plexus is well developed, the nerve may be surrounded by blood spaces from the point where it enters the dura until it leaves the cavernous sinus to pass into the orbit. In such an instance, the nerve, as it passes through Dorello's canal, runs in the middle of the inferior petrosal sinus. In one specimen, in which the suture was wide, the nerve occupied a position between the petrous tip and the sphenoid (fig. 14). In such a situation a slight blow applied to the region of the mastoid might easily injure it, or a small amount of edema could compress it.

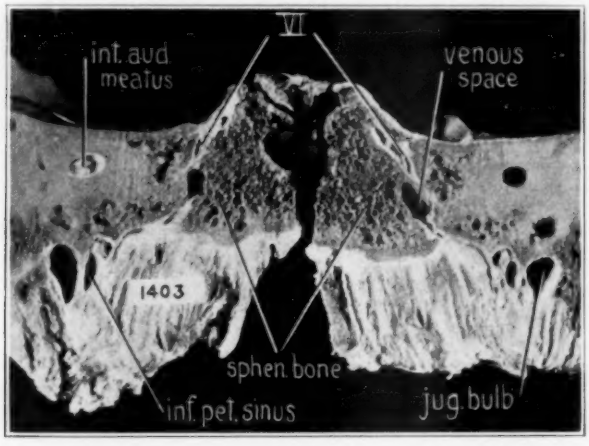

Fig. 14.—A vertical section of the left temporal bone, showing the abducens nerve deep in the gap between the apex and the side of the sphenoid. Its position is vulnerable in the event of edema or of a blow on the side of the head. A large opening is seen just below the nerve, which is a venous space and connects with the inferior petrosal sinus above and the petro-occipital sinus below. The section is cut in plane *A* (fig. 1).

Occasionally the sphenoid sinus extends well back into the basal part of the body of the bone, and the nerve, piercing the dura near the midline, makes an impression on the posterolateral wall of the sinus, bringing it into relation with possible influences arising from within the sphenoid sinus. Often the nerve is enclosed in a heavy, unyielding dural sheath for a considerable distance, in which case a small amount of edema could cause an interruption of function. In some specimens the nerve pierces the dura lateral to the inferior petrosal sinus and proceeds upward over the petrous apex without coming into relation with the blood spaces until it enters the cavernous sinus.

Like the mastoid, the interior of the petrous apex varies over a wide range, thereby making each individual problem a study in itself (fig. 15). The two may be different in the same person, although there is a tendency toward symmetry in their construction. The petrous apex may be described as noncellular or sclerotic, when cancellous bone has never developed (fig. 16), or as cellular, when the entire bone is filled with either diploic structure or pneumatic spaces. Since diploe must precede pneumatization, both are usually present. Marrow spaces, air cells and sclerotic bone may then be found in the same specimen; the pneumatic type may consist of large cells, small cells or a mixture of the two. When the cells are large, the cortex tends to be thin, at times

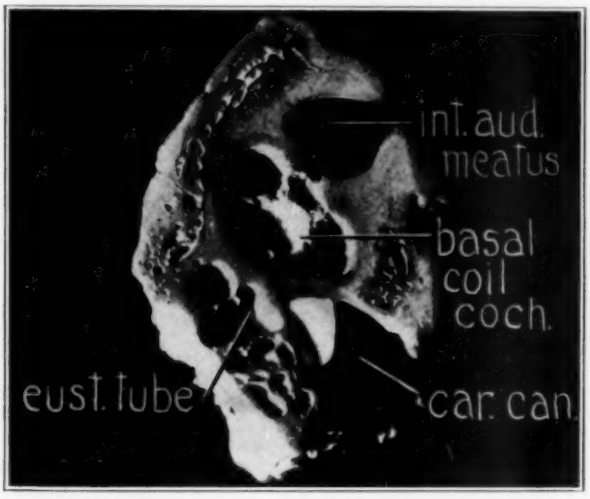

Fig. 15.—A transverse section of the petrous apex of the left temporal bone from the skull shown in figure 3. Cell tracts extending into the apex are located above the labyrinth along the cerebral surface, below the labyrinth and internal auditory canal along the cerebellar surface and below the eustachian tube along the lower, or extracranial, surface. The section is cut in plane *C* (fig. 1).

no thicker than tissue paper, and readily perforated by pathologic processes. The smaller cells are likely to be surrounded on two or three sides by compact bone, with a thick cortex, more resistant to the progress of infection. Pneumatization thus has a distinct bearing on the symptoms and outcome of petrous disease. The factors which determine the extent and direction of pneumatization are obscure, but there is a tendency for the process to continue more or less throughout life. However, bones in which air cells are normally found may be pneumatized to the limit in any or all directions, and the process may extend across sutures into adjoining bones if fusion of the suture is complete

early in development. The petrous apex, then, may be completely pneumatized (figs. 17 and 18), and such pneumatization may extend across the petrobasilar suture into the sphenoid and the occipital bone. Occasionally a tier of cells is found between the posterior semicircular canal and the cerebellar wall, in which case the endolymphatic duct extends across the field of operation and may easily be severed, with danger of infecting the labyrinth, or Ménière's syndrome may result from inadequate drainage of the endolymph.

On account of the great variation in the location of pneumatic spaces in the petrous apex, it would seem that no operation yet devised is applicable in every instance of suppuration in this area. Certain speci-

Fig. 16.—A horizontal section of the left petrous bone, showing a sclerotic apex. A probe is in the eustachian tube. The section is cut in plane *B* (fig. 1).

mens indicate clearly that without entering the tympanic cavity one may expose all but the front part of the labyrinthine capsule and that the apex may be entered by following the subarcuate canal through the arch of the superior semicircular canal (Frenckner's operation). Also the extreme depth of the apex may be reached by following the cell tracts along the cerebellar wall between the jugular bulb below and the posterior semicircular canal above. The surgeon, therefore, should be familiar with the various possibilities and apply his knowledge and skill so as to meet the problem at hand. In the interpretation of roentgenograms one must bear in mind the possibility of rather large venous spaces along the carotid canal, which might be mistaken for air cells. There may be present also a large cleft along the petrobasilar suture,

which dips into the apex between the carotid canal and the cerebellar surface. In life these spaces are filled with venous blood. Their walls are more irregular in outline than are those of pneumatic cells. The density of osseous tissue increases with age; therefore, one must remember that the bones of children are more transparent than those of adults.

Pain is a frequent and important symptom of inflammation of the petrous apex; yet it is not a certain indication of the severity or progress

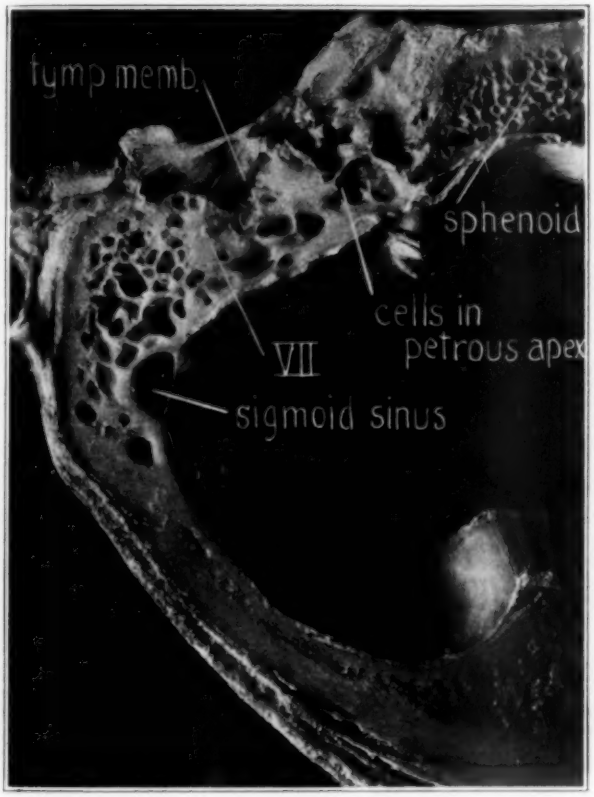

Fig. 17.—A horizontal section, showing large pneumatic cells extending to the limit of the apex. Note the proximity to the diploe of the sphenoid. The section is cut in plane *B* (fig. 1).

of the pathologic process, as Friesner [5] has shown from his microscopic studies, nor does it locate definitely the area involved. After sectioning the petrous pyramid of 24 subjects who had died of intracranial complications in which the petrosa was diseased, he said: "Even in retro-

5. Friesner, I.; Druss, J. G.; Rosenwasser, H., and Rosen, S.: Suppuration of the Petrous Pyramid, Arch. Otolaryng. **22**:659 (Dec.) 1935.

spect, it is at times difficult or impossible to correlate the clinical with the histologic observations." In many instances his studies gave evidence of disease lateral to the apex, with the latter showing little or no involvement, and yet the symptoms were identical with those which occurred when the apex was involved. He further stated that, "provided other etiologic factors have been excluded, the presence of pain is much more important than its localization."

The mechanism by which intracranial pain is produced and distributed is not clear. Instances have been reported by Brunner [6] and

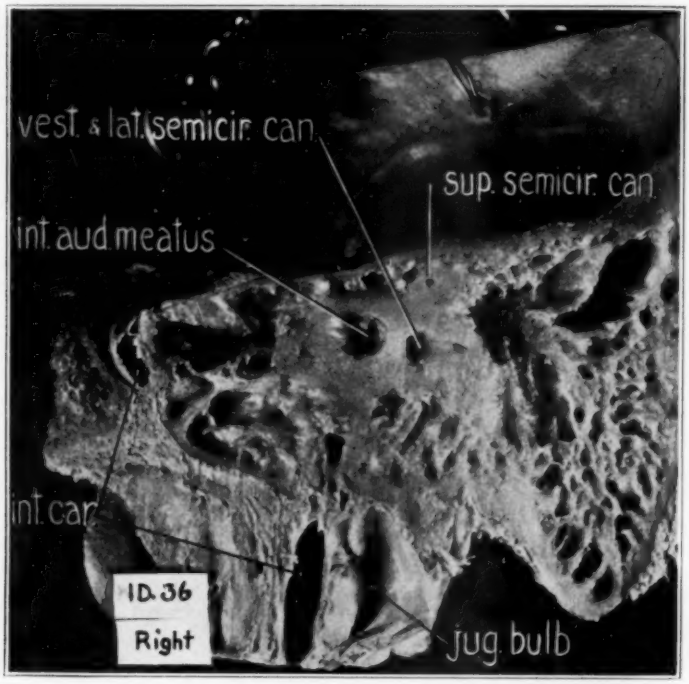

Fig. 18.—A vertical section of the right temporal bone, showing extensive pneumatization of the apex. Cell tracts are seen above and below the labyrinthine capsule. The section is cut in plane *A* (fig. 1).

others [7] in which the gasserian ganglion showed extensive disease but pain was not a part of the clinical picture. Pain in the head can be

6. Brunner, H.: Ueber eitrige Erkrankungen an der Felsenbeinspitze, Ztschr. f. Hals-, Nasen- u. Ohrenh. **25**:383, 1930.

7. Uffenorde, W.: Eine zu vielfachen Verwicklungen führende, sehr ausgedehnte perilabyrinthäre Eiterung im histologischen Bilde, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **122**:288, 1929. Hilgerman, R.: Die Beteiligung des Ganglion Gasseri bei Mittelohreiterungem, Ztschr. f. Ohrenh. **40**:311, 1901-1902.

produced by either increasing or decreasing the intracranial pressure. Discussing headache, Best and Taylor [8] said:

> The sensitivity of the pial vessels has led to the belief that the majority of headaches are of vascular origin, the pain being due to vasodilatation and the excitation of the sensitive nerve endings situated upon the vessel wall or in its immediate neighborhood.

It would seem, then, that pain is not dependent on involvement of the nerve trunk, or ganglion, but rather on the irritation of the nerve endings which may be located near or at some distance from the ganglion, or nerve trunk, in question. These minute nerve endings are probably connected with the autonomic nervous system; so the pain of petrous disease may be initiated anywhere along the carotid sympathetic plexus or perhaps along the petrosal nerves and be referred to any point over the wide distribution of the trigeminal nerve, thus producing, for example, toothache or orbital pain, as well as the Horner syndrome. The recurrent branch of the mandibular nerve which accompanies the middle meningeal artery may easily be affected at the foramen spinosum, on account of its proximity to the petrosa, and give rise to pain in the parietal region. The glossopharyngeal, the vagus and the spinal accessory nerves are in relation to the pyramid at the jugular foramen, and, if all are affected, may produce Vernet's syndrome.

SUMMARY

Individual susceptibilities and anatomic variations are common and must be taken into account in the evaluation of signs and symptoms accompanying infection of the petrous apex.

The temporal bone is unique in its connection with other bones. Provision is made for movement and growth with minimum stress on or shock to the hearing apparatus.

The petrous portion of the temporal bone has two intracranial and one extracranial surface, all of which are in communication with each other through the venous spaces at the mesial end. The cerebral and the cerebellar surfaces are separated by the firm attachment of the tentorium cerebelli along the superior border of the pyramid, except at the mesial end.

The petrous apex is that portion of the pyramid mesial to the labyrinthine capsule. An imaginary line beginning at the notch for the exit of the cochlear duct and ending at the hiatus Fallopii would mark its lateral boundary.

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8. Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice, Baltimore, William Wood & Company, 1937, p. 1445.

In connection with the cerebral surface of the petrous apex, it is important to remember its relation to the temporal lobe, the greater and the lesser superficial petrosal nerves and their foramens and to the foramen for a branch of the middle meningeal artery; its impression for the gasserian ganglion, where the bone is thin and perforated by small openings for the transmission of veins in about 75 per cent of specimens examined; its relation to the foramen lacerum for the internal carotid artery, the pericarotid venous plexus and the carotid sympathetic plexus; its proximity to the foramen spinosum for the middle meningeal artery and vein and a recurrent branch of the sensory portion of the mandibular nerve, which may account for parietal pain, and its relation to the foramen ovale for the mandibular nerve and the venous connections between the cavernous sinus and the pterygoid plexus.

In connection with the cerebellar surface of the petrous apex it is important to remember its relation to the pons, medulla and cerebellum, to the abducens nerve, to the internal auditory meatus for the facial and the auditory nerve and the auditory artery and vein, to the hiatus cochlearis for the cochlear duct and an artery and vein and to the groove for the inferior petrosal sinus, and its proximity to the subarcuate fossa for the passage of a small artery and vein and to the jugular bulb, the glossopharyngeal and the spinal accessory nerves.

In connection with the extracranial surface of the petrous apex its location at the upper end of the carotid sheath must be kept in mind, as well as its relation to the eustachian tube, the groove for the sinus of Englich and the carotid canal. It is perforated for the passage of small veins and is situated close to the pharyngomaxillary space, the lateral pharyngeal recess and the retropharyngeal space.

The abducens nerve is placed in a vulnerable position on account of its intimate relation to venous spaces throughout most of its long course, also on account of its relations to the basisphenoid and the petrous apex.

The petrobasilar suture may be fused with the diploic structure of the petrosa continuous with that of the sphenoid and occipital bones. Thus osteomyelitis may extend in either direction.

The process of pneumatization seems to follow the formation of the diploe; therefore, cancellous structure is usually found along with the air spaces. Thus osteomyelitis and osteitis are often present at the same time.

Venous spaces along the internal carotid artery and the petrobasilar suture may on the roentgenogram be mistaken for pneumatic cells.

The Gradenigo syndrome is often not complete and therefore should not be alone relied on for diagnosis or therapy.

ABSTRACT OF DISCUSSION

DR. J. PARSONS SCHAEFFER, Philadelphia: A study such as this raises many important questions, some specific, others general. The basic structure and relations have been covered by Dr. Mellinger, and little can be added to this phase of the subject. Reference was made to human constitution. This aspect is intriguing and complex. The problem from this point of view is often baffling; true evaluations and interpretations are difficult. Of the several factors involved, structure is extremely important. The problem of anatomic variation was stressed, and rightly so. It is not merely of academic interest; it must often have a profound bearing in practical work as well. There are normally great variations in the character of the cancellous or diploic bone, which is in some cases coarse and almost cellular and in other cases extremely fine. Roentgen interpretation is at times difficult, if not impossible, especially in early osteomyelitis. The clinical symptoms and signs are highly important; the otologist and the roentgenologist must coordinate their efforts. The petrous apex may contain a number of large cells or, sometimes, a large single cell. The arrangement of such cells is not constant. Although the petrous bone is usually separated from the basilar portion of the occipital bone and the extreme dorsal portion of the sphenoid by a suture, it is sometimes coossified with these bones by true synostosis, and when the sphenoid sinus has hollowed out the body of the sphenoid to an extreme degree, penetrating even into the basilar process of the occipital bone, the sinus may be a factor in infecting the cancellous bone or cells in the petrous apex, especially when the diploic spaces of the bones are continuous. The sphenoid sinuses are extremely variable and usually asymmetric. It is possible from an anatomic point of view for both canals of Dorello to be in contact with a sphenoid sinus of one side. Involvement of the nervous system in disease of the petrous tip cannot be constant in its manifestations because of the varied structure.

Morphologic individualism is an important aspect of the human constitution. Although one must, perhaps, approach clinical problems involving morphology from the point of view of the so-called typical, one assumes grave responsibilities in doing so. Mistakes may be made because variations in important and essential details go unrecognized or are deliberately ignored. From the point of view of the variations of the intrinsic structure, it would appear illogical to assume that any one procedure or technic can be accepted as a standard method to be used in all cases in an effort at eradication of disease in the petrous part of the temporal bone.

DR. OLOF LARSELL, Portland, Ore.: As I studied Dr. Mellinger's preparations and noted the thoroughness with which he had dissected and sectioned the various planes of the temporal bone and its petrous apex, I realized how little is ordinarily taught of the features of anatomy with which the surgeon must deal. Dr. Mellinger's paper gives a clear description of the features shown by his dissections.

Dr. Mellinger has performed a distinct service in pointing out the marked variations in the internal structure of the petrous bone. These variations make each case present an individual anatomic problem for the surgeon, which can only be analyzed as it is also being solved during operation. The paper emphasizes the need of broad knowledge and experience.

The comment I would offer pertains to the relations of the several nerves and the possibility, by detailed preliminary study, of localizing the particular part of the petrous bone involved in each individual case. Dr. Mellinger has pointed out that the danger of confusing venous spaces with pneumatic spaces in roentgen examination makes this method not reliable.

Since there are important relations of the petrous bone to the gasserian ganglion, which extends into an impression near the tip of the petrous bone, the floor of which is perforated by small openings from the bone, there are the following possibilities: (1) The trigeminus may be involved, by pressure if not by actual infection, which may yield some sign, from trigeminal symptoms, that the infection extends to the tip of the petrous bone; (2) the abducens nerve may be involved, as has been recognized. In one of Dr. Mellinger's dissections, which I examined with him, a nerve trunk, as it appeared, arose from the internal carotid plexus and joined with the sixth nerve. If this was made up of cervical sympathetic fibers, as appears likely, it can be regarded as an aberrant bundle, reaching the cranium by the root of the sixth nerve instead of by its usual course. If it were involved with the sixth nerve as it passes through or near Dorello's canal, one can see possibilities of some aspects of Horner's syndrome in addition to symptoms of involvement of the abducens nerve.

Possibilities of involvement of the facial nerve in the facial canal are obvious if the basal region of the petrous bone is involved. It appears to me not only that signals of pain, such as Gradenigo's syndrome of violent temporoparietal pain, should be looked for but that close attention may show various indications of motor muscular and secretory disturbances due to involvement of the various nerves which pass in such close relation to various parts of the petrous bone.

I wish to congratulate Dr. Mellinger on an excellent and valuable piece of anatomic work, which should have important clinical implications. The practicing otologist can supplement it by accurate observations of disturbances of sensory, motor and secretory nerves.

Dr. Louis K. Guggenheim, Los Angeles: There is a point in Dr. Mellinger's splendid presentation which should be discussed—the question of bilateral involvement of the sixth nerve. In 1 case a man of 22 with petrositis showed involvement of the sixth nerve on the affected side and, several weeks later, involvement of the opposite nerve.

The temptation in such a case is to conclude that basilar meningitis is present, but I now believe that in many cases of this type the condition is probably due to simple edema. This patient made a complete recovery, including return of function of both sixth nerves.

About three months ago I was asked to see a woman of 32 who had symptoms of petrositis and also intracranial involvement, with bilateral involvement of the sixth nerve. The physician in charge had concluded that basilar meningitis must be present and that possibly there was a localized abscess which could be reached through the posterior wall of the pharynx, as in 1 case of Ferris Smith's. I insisted that the operation on the ear had not been finished (a simple exenteration had been done), and the surgeon felt that he had done everything that could possibly be done to the aural region. I asked that a revision of the mastoid be done. He performed the Ferris Smith operation through the posterior wall of the pharynx and found nothing; the patient died. There was a temporosphenoid abscess on the affected side and only basilar edema.

Now, the point is that a bilateral involvement of the sixth nerve does not necessarily mean basilar meningitis or a localized abscess, but probably in most instances simple edema.

Dr. Joseph C. Beck, Chicago: I had the pleasure of being seated at the table for some time with Dr. Mellinger and seeing his exhibit, and, like the other members, I admired the work; I am sure it is a great step forward to have these exhibits before a paper is read.

I want to present an instrument, because it fits in here in the discussion. It is an instrument used in obtaining a preparation, particularly in cases in which one is not permitted to do a postmortem examination, yet probably would be permitted to remove some of the tissues.

I have worked on the problem of obtaining the greater part of the temporal bone—not the entire temporal bone but all that one is interested in, namely, the mastoid, the middle ear and the petrous pyramid. I am not at this time going into the technic of using the trephine; I shall save that for another occasion—I merely wish to show the instrument in connection with Dr. Mellinger's paper and to put it on record. One really obtains not only the petrous pyramid but the internal ear, which is of interest in many conditions. I have named the instrument a "corer."

Dr. William J. Mellinger, Santa Barbara, Calif.: Since there are no questions to answer, I can only emphasize a few points which seem pertinent in view of this study.

On account of the great variation in the cellular arrangement, one should not approach the petrous apex surgically with a preconceived notion of doing somebody's operation but rather follow the cell tracts, adjusting the technic so as to conform to the individual anatomy of the subject at hand. Many specimens show clearly that by extending the simple procedure for operation on the mastoid along the posterior wall the very depths of the apex may be reached; then, if further surgical intervention is indicated, the operation may be converted into a radical one at some future time. The surgeon's duty is to bring about a cure with a minimum of functional disturbance.

The ductus endolymphaticus is in danger of injury when cells are found between the posterior semicircular canal and the cerebellar wall. Thus infection might extend to the labyrinth or Ménière's syndrome develop from interference with the endolymphatic drainage.

A STUDY IN PRESBYCUSIS

AUDITORY LOSS WITH INCREASING AGE AND ITS EFFECT ON THE PERCEPTION OF MUSIC AND SPEECH

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This study is an attempt to investigate three problems: First, what loss in auditory acuity, independent of special pathologic conditions, accompanies increasing age? Second, how does such auditory loss affect what one hears in a musical tone? Third, how does such a loss affect one's perception of speech?

I. AUDITORY ACUITY AND AGE

Previous studies[1] and ordinary observation have indicated that a person progressively loses his hearing as he grows older. This phenomenon is known as "presbycusis." The first problem in these investigations is concerned with the extent and the nature of presbycusis in persons above 50.

The auditory sensitivity of 168 ears was measured by means of a Western Electric 2-A audiometer, which produces eight frequencies in octave intervals ranging from 64 to 8192 cycles. Both ears were tested for 80 subjects and only one for 8. Sixty-four subjects received an otologic examination to insure that there were no indications of previous pathologic changes in the ear that might account for the present status of hearing. Those who did not receive a special medical examination reported that, to their knowledge, they had had no previous aural trouble. Their audiograms indicated auditory sensitivity typical of their age. The measurements of auditory acuity were made in a sound-proof room in the psychologic laboratories at the State University of Iowa, and the otologic examinations were made in the department of otolaryngology at University Hospital, Iowa City.[2]

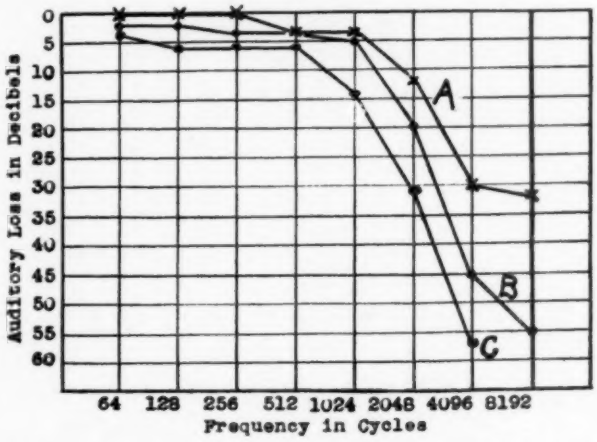
The experimentation reported in this paper was conducted while I was an Eastman Research Fellow in the Psychological Laboratories of the State University of Iowa and made possible by the Eastman Foundation and Dean Carl E. Seashore.

1. (*a*) Bunch, C. C.: Age Variations in Auditory Acuity, Arch. Otolaryng. **9**:625 (June) 1929. (*b*) Montgomery, H. C.: Do Our Ears Grow Old? Bell Lab. Rec. **10**:311, 1932.

2. Dr. D. M. Lierle, head of the department of otolaryngology and oral surgery, University Hospital, cooperated in the investigation.

In arranging the data from the measurements indicated the subjects were grouped into the following four age ranges: (1) 50 to 59, (2) 60 to 69, (3) 70 to 79 and (4) 80 to 86. The first group included 60 ears; the second, 80 ears; the third, 22 ears, and the fourth, 6 ears.

The mean auditory thresholds of the subjects, grouped according to the four age ranges suggested, for the eight frequencies are presented in table 1 and represented graphically in the chart. The mean auditory threshold is expressed in decibels above an arbitrary reference point of zero, which represents normal hearing on the calibration of the 2-A audiometer. The mean thresholds given in the table indicate loss of hearing in decibels. The values in parentheses inserted to the right of those for the mean auditory threshold in table 1 represent the average deviation of the individual measures from the mean.



Mean auditory loss with age. *A*, age 50 to 59; *B*, age 60 to 69; *C*, age 70 to 79.

An examination of the data presented in table 1 reveals the following significant facts:

1. Up to the age of 70 hearing remains practically normal for frequencies up to 512 cycles. In the age range 70 to 79 the loss for low frequencies is slight.

2. The loss for 1024 cycles is small until one reaches the age range from 70 to 79, in which the loss is only 14 decibels.

3. In subjects of 50 presbycusis is already apparent for frequencies of 2048 cycles and beyond. The higher the frequency the greater is the auditory loss.

4. Looking across the table horizontally at the mean values for frequencies 2048, 4096, and 8192, one notes that presbycusis for the high frequencies becomes progressive with increasing age.

5. Most persons above 60 have lost their serviceable hearing for frequencies above 4096 cycles.

6. The variation of the individual measures from the mean is greater for frequencies above 2048 cycles than for those of 1024 cycles and lower.

Only 3 subjects in the group were over 80. The measurements may be considered suggestive but cannot be taken as representative of auditory acuity in the advanced years.

There was no significant difference between the two ears of a given subject. In the majority of cases the hearing curves were approximately the same for the two ears. In cases of slight variation, one ear might be from 5 to 10 decibels superior at one frequency and inferior by a similar amount at another frequency.

TABLE 1.—*Mean Auditory Thresholds According to Age*

Frequency in Cycles	Age in Years			
	50-59	60-69	70-79	80-86
64	0 (3)	2 (4)	3 (6)	12 (5)
128	0 (3)	2 (5)	6 (6)	15 (5)
256	0 (3)	3 (5)	6 (6)	13 (3)
512	3 (4)	3 (5)	6 (6)	8 (5)
1024	3 (5)	5 (5)	14 (9)	8 (7)
2048	12 (9)	20 (11)	31 (16)	27 (14)
4096	29 (13)	45 (18)	57 (17)	60 (15)
8192	31 (16)	55*	.....	58 (5)

* This is the median value. In the age range from 60 to 79 only 56 of the 102 ears heard the 8192 cycle tone at the maximum output of the audiometer, which was 60 decibels above the normal threshold of that tone.

This investigation did not include any subjects whom diagnosis or medical history revealed to have had previous involvement of the middle ear. The otologic examinations revealed that most of the subjects had some pathologic condition in the upper part of the respiratory tract; however, many whose diagnoses showed chronic disease of the nose and throat, deflected septums or other disturbances possessed greater acuity of hearing than those who evidenced only minor abnormalities in these areas. Bunch [3] reported that loss of hearing in patients with arteriosclerosis, hypertension, chronic cardiac diseases, syphilis or malignant conditions is not greater than in aging persons who are not recognized to have any of these disturbances. There is reason to believe that the auditory losses reported in table 1 are due to increasing age and not to any special pathologic conditions external to the inner ear.

Two previous studies have reported variations in auditory sensitivity in relation to age. Bunch [1a] conducted an extensive investigation of the problem in the Otological Research Laboratory of Johns Hopkins Uni-

3. Bunch, C. C.: Further Observations on Age Variations in Auditory Acuity, Arch. Otolaryng. **13**:170 (Feb.) 1931.

versity. His subjects were classified into age groups by decades, the first decade beginning at 20. Only the measurements for the last two decades, from 50 to 70, can be compared with the data presented in this study. Bunch reported loss of hearing for the low frequencies (up to 512 cycles) varying from 19 to 28 decibels. The loss reported in this study for the same frequencies varies from 2 to 6 decibels. The auditory loss reported by Bunch for the 1024 cycle tone is much greater than that found in this study. Bunch stated: "The tests were conducted in a small room set apart for that purpose or in hospital wards. Conditions were not ideal, for neither the testing room nor the wards were free from disturbing noises." Evidently the values which he gave for this low frequency range represented measurement of room noise rather than auditory loss. The losses reported for the 2048 cycle tone were 27 and 38 decibels for the two decades beginning at 50 and at 60. The values presented in this study are 12 and 20 decibels. The values reported by Bunch for the tones of 4096 and 8192 cycles are comparable to those reported from this investigation.

Montgomery [1b] reported the results of a similar investigation made at the Bell Telephone Laboratories. The age of his subjects ranged from 20 to 60. Only measurements for the decade from 50 to 59 inclusive can be compared with data from this study. Such a comparison reveals close agreement between the two sets of measurements. Both studies present evidence that hearing remains practically normal for the low frequencies, with presbycusis progressing with increasing age, and that the higher the frequency of the stimulating tone the greater is the presbycusis. This study presents data for the ages from 60 to 79, two decades beyond the ages investigated by Montgomery.

II. PRESBYCUSIS AND THE PERCEPTION OF MUSIC

The second division of this study attempted to determine to what extent a loss of auditory acuity due to increasing age modifies the quality of a musical tone. Seven subjects with normal hearing for all frequencies from 64 to 8192 cycles and 10 with a loss of sensitivity for the higher frequencies were used in this experimentation. The hearing curves for these 10 subjects were representative selections from the curves for the presbycusic subjects used in the first division of the study. Each of the presbycusic subjects had normal hearing for tones from 64 to 512 cycles. At 1024 cycles there was a loss of only a few decibels, increasing for tones from 2048 to 8192 cycles.

Apparatus and Procedure.—A violin tone produced by a continuously vibrating A string was transmitted to the observers by an amplifying system of high quality. This system, which I used in a previous clinical study in audition,[4] consisted of

4. Kelley, N. H.: A Comparative Study of the Response of Normal and Pathological Ears to Speech Sounds, J. Exper. Psychol. **21**:342, 1937.

a Western Electric nondirectional dynamic microphone no. 630 A, a preamplifier, a power amplifier and a Western Electric moving coil or dynamic receiver no. D 90794. The microphone and preamplifier were operated in a quiet room; the power amplifier and receiver were located in a neighboring sound-proof room. The intensity of the transmitted sound was controlled by means of accurate attenuators calibrated directly in decibels. Frequent check showed that the gain and stability of the system remained constant. The response of the amplifiers was uniform up to 13,000 cycles.

Connected with the amplifying system was a filtering circuit of three units. This circuit consisted of three General Radio low pass filters, type 830, with cutoffs at 2000, 4000 and 6000 cycles. Each filter was controlled by an individual switch, which enabled the experimenter to insert it or exclude it from the sound transmission system silently and quickly. There was no insertion loss or decrease in intensity of the presented tone when a filter was inserted.

The violin string was set into vibration mechanically. The apparatus [5] consisted of a violin stand and a revolving bow hair device. The latter was adjustable so that the bow hair could be applied to or drawn away from the violin string. Thus, the pressure exerted by the bow hair on the string could be varied under control. The bowing device was driven by an alternating current motor, and the violin was securely fastened within the framework of the stand.

By attaching a telegraph key switch across an attenuator, it was possible to present through the receiver a tone of any desired duration. Each subject was thus presented with a series of paired tones. One of the pair was the natural unfiltered tone, carrying the high frequency components. The other member of the pair was the same tone filtered by one of the low pass filtering units. The position of the natural full tone was varied in the series, and each subject was asked to judge on the basis of a difference in the quality of the two tones whether it came first or second in the pair, with an attitude toward detecting the presence of the higher partials in the violin tone. If a difference was detected, the intensity of the tone was decreased by varying steps until a threshold of discrimination was reached. This threshold is defined as the lowest intensity about the auditory threshold of the observer for the unfiltered tone at which 75 per cent of the judgments were correct. Only monaural hearing was used.

The thresholds of discrimination for persons with normal and with presbycusic hearing are presented in table 2.

The range of high frequency components eliminated from the complex violin tone is greater the lower the cutoff frequency of a low pass filter. Hence it should be easier to detect a change in the quality of the tone with the insertion of the 2000 cycle low pass filter than with the insertion of the 4000 or the 6000 cycle filter. In other words, the lower the cutoff frequency, the lower should be the threshold of discrimination. The data presented in table 2 indicate that this is true both for persons with normal and for those with presbycusic hearing. With the insertion of the 2000 cycle filter, the observer with normal hearing can judge accurately a difference in the quality of the given violin tone at an intensity as low as 2 decibels above his auditory threshold. With the

5. This apparatus was designed by Dr. Arnold Small and has been used by him in conducting studies in tone quality.

insertion of the 4000 cycle filter, the lowest intensity above threshold at which a difference in tonal quality could be detected varied from 4 to 24 decibels. For example, subject 5 could make no discrimination between the filtered and the unfiltered tone at intensities less than 16 decibels above his threshold. With the 6000 cycle filter, the threshold of discrimination is higher for each subject than with the 4000 cycle filter. In all cases, however, the subject with normal hearing perceived a difference in tonal quality when the higher frequency partials were eliminated from the complex violin tone.

The data secured from the persons with presbycusic ears are markedly different from those obtained from subjects with normal hearing. With the insertion of the 6000 cycle filter, there was no discrimination of any

TABLE 2.—*Thresholds of Discrimination*

	Subject	Low Pass Filter 2,000	4,000	6,000
1. Normal ears	1	2	4	24
	2	2	8	20
	3	2	10	42
	4	2	11	19
	5	2	16	20
	6	2	17	20
	7	2	24	42
2. Presbycusic ears*	1	6	52	
	2	4	..	
	3	6	56	
	4	..	..	
	5	..	..	
	6	16	..	
	7	24	44	
	8	..	..	
	9	10	..	
	10	..	..	

* The absence of values for presbycusic ears indicates that the observer was unable to detect any qualitative difference between the natural and the filtered tone at any intensity.

change in the quality of the tone. With the insertion of the 4000 cycle filter, only 3 of the 10 subjects detected a difference in tonal quality. These discriminations occurred only when the intensity of the stimulating tone was 44, 52 and 56 decibels above auditory threshold. Six of the subjects judged a difference in tonal quality with the insertion of the 2000 cycle filter. The thresholds of discrimination for these 6 are higher, however, than for persons with normal hearing. These data indicate that the elimination of frequencies 4000 cycles and above does not affect the experienced quality of a violin tone when heard by a person with presbycusic hearing. This may be interpreted to mean that a person with the decreasing sensitivity for the higher frequencies which accompanies increasing age does not experience the same quality in a musical tone that one does who has normal hearing. In other words, one's experience of an orchestral selection at 70 does not contain the same richness

of tonal quality as it did when one was younger and had normal hearing for the high frequencies. Since the development of presbycusis is gradual, the change in experienced quality of tone in music is probably unnoticed by many. In the case of trained musicians, it is possible that subjective experience, in terms of dynamic gestalten, might compensate for those higher partials present in the physical stimulus which produce no response in the mechanisms of hearing. All persons would be able to appreciate melody and rhythm. To persons with presbycusis, complex tones would lack brilliance, and they would not detect qualitative differences between musical instruments as they did when younger.

III. PRESBYCUSIS AND THE PERCEPTION OF SPEECH

The third phase of the experimentation attempted to study the effect of presbycusis on the articulatory recognition of vowels and consonants. Eight subjects with normal hearing and 12 with presbycusic ears were used in this study. Speech was transmitted over the amplifying system

TABLE 3.—*Percentage of Speech Sounds Recognized*

	Vowels				Consonants			
Decibels above threshold	40	30	20	10	40	30	20	10
Normal ears	99	99	99	94	98	97	89	65
Presbycusic ears	92	90	79	49	75	63	45	21
Mean deviation								
Normal ears	2	1	2	4	1	2	4	11
Presbycusic ears	4	6	12	26	9	13	16	16

mentioned in part II. Measurements were made at 40, 30, 20 and 10 decibels above the average normal threshold of hearing with a test list of seventy-five monosyllabic words. Several lists were used, each containing the same words but in a different arrangement. One sound only was graded in each word. Each list contains twenty-five vowels and fifty consonants. Two lists, or one hundred and fifty words, were presented at each intensity. The data on articulatory recognition of speech are presented in table 3 in terms of the percentage of sounds recognized.[6]

The average person with normal hearing prefers to listen to continuous discourse at an intensity 38 decibels above threshold.[4] At this preferred intensity, persons with presbycusic hearing recognize vowels almost as well as do persons with normal hearing, but in the recognition of consonants they are inferior at all intensities. This is to be expected, since the consonants are complex sounds containing high frequency com-

6. In a previous study[4] I investigated articulatory recognition of speech sounds in persons with conduction deafness, who had approximately equal losses of hearing measured in decibels over their range of audible frequencies.

ponents which elicit no response in the mechanism of the presbycusic ear. Recognition of consonants is essential to understanding the spoken word. Hence, persons over 60 years of age frequently report that they have difficulty in understanding a person who speaks across the room, from an adjoining room or in a noisy environment. In the recognition of vowels the presbycusic ear is not seriously handicapped except at low intensities.

CONCLUSIONS

1. Hearing for the frequencies up to 1024 cycles remains practically normal with increasing age up to 70. Between 70 and 80 the auditory loss at 1024 cycles is 14 decibels.

2. At 50, presbycusis is already apparent for frequencies of 2048 cycles and beyond. The higher the frequency the greater is the auditory loss.

3. Presbycusis for the high frequencies progresses with increasing age.

4. The elimination of all frequencies above 4000 cycles does not affect the experienced quality of a violin tone when heard by a person over 60 who manifests presbycusis. Such elimination definitely alters the character of the same tone heard in the normal ear.

5. In the recognition of vowels the average person past 60 is not seriously handicapped except at a low intensity, 10 decibels above threshold.

6. In the recognition of consonants a person past 60 is inferior at all intensities to a person with normal hearing. At the intensity preferred by a person with normal hearing for ordinary conversation (38 decibels) the presbycusic person recognizes 75 per cent of the consonants.

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HISTORY OF AUDIOMETRY

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The story of the development of audiometry through successive stages not only is historically interesting but throws light on problems met and overcome.

For many years there were in desultory use various devices capable of producing sounds of controlled intensity. The first types were clock-like, giving off air-borne sound to the tubes of a stethoscope; the sound distributor head had a valve which could be gradually closed. Another model used a tripped hammer to strike a metal rod and produce the testing sound; in another a tuning fork was struck. Later, with the advent of simple electric circuits, came the use of the buzzer type of audiometer, in which the bell announcer circuit with electromagnet and interrupting armature was used.[1] The electric interruptions were sent out to a telephone receiver, and the attenuation was produced either by a resistance in the telephone line or by cutting down the battery power to the buzzer. Some mechanically minded otologists adapted the faradic machine for the same purpose, with the secondary coil pulled in or out to change the intensity of the noise; others used the circuit and attenuation of the direct current wall panel.

The first attempt at standardizing these heterogeneous instruments was the invention of the buzzer audiometer by the Western Electric Company. This machine produced a mixed tone or noise, which was attenuated by a rheostat graduated in definite electrical units of intensity. It was operated by a battery, was heavy and produced such vibration in the floor that it was often difficult to tell whether the deafened person was reporting the feeling or the hearing.

At that point I adapted the cycle of the street or house current to give a hum on a telephone receiver.[2] Attenuation was easily accom-

1. The following instruments are representative: M. J. Haffey's otometer, presented before the Sections on Ophthalmology and Otolaryngology of the Academy of Medicine of Toronto, in October, 1924; the Pilling-McCallie audiometer, manufactured by George P. Pilling & Son Co., Philadelphia, in 1911, and F. D. Kranz' audiometer, made at Riverbank Laboratories, Geneva, Ill. No published literature on these audiometers is available; they were experimental efforts and are not on the market.

2. Macfarlan, D.: Circuit Plans for Inexpensive Audiometers, Arch. Otolaryng. **7**:527 (May) 1928.

plished by variable resistances. (The method no doubt occurred to many otologists and others almost simultaneously; it is still useful in the making of an inexpensive noise producer for dampening, and Dr. Guild and I use it for this purpose, for which it was recommended by the committee of the American Otological Society in 1937.)

The growing experience with testing hearing with noise as a stimulus proved it to be not altogether satisfactory. No two instruments produced identical noises, and none except the Western Electric buzzer had an intensity control based on a scientific unit of measurement. The noises were an unknown mixture of frequencies, the use of which gave no clue as to the patients' hearing in various parts of the auditory range. Testing with noise gave little information as to a patient's ability to hear speech, and educators as well as afflicted persons were interested in how well language was heard. (It is still impossible to correlate testing with single tones and testing for hearing of speech.) Testing across the frequency range was desired, and there was a definite need for a more scientific test of hearing of speech. (The old test with the whispered voice was recognized as unsatisfactory, since intensities were variable.)

TESTS FOR HEARING OF SPEECH

In 1904, Dr. Sohier-Bryant had presented before the American Otological Society [3] a phonograph audiometer in which speech was used as the testing stimulus. The idea was perhaps a natural consequence of Edison's invention of the wax cylinder recording and reproducing device. Unfortunately, no model of Dr. Bryant's machine has been preserved, but the illustrations in his original paper show it to be Edison's phonograph, with speech put out through stethoscopic tubes and intensity controlled by a valve.

Several years elapsed before the appearance of the "audiophone," a phonograph with an electric pick-up and with speech sent through a telephone receiver. By the newer method it was readily possible to control intensities by resistances. Professor Bristol, of Waterbury, Conn., was responsible for the invention, and Harris Taylor and Miss Berry found use for it in the Lexington Avenue School for the Deaf. I have still in use in a clinic for the prevention of deafness one of the early Bristol machines, to which has been added a decibel meter.

In 1920 appeared Carl E. Seashore's studies [4] on the testing of musical talent. The instrument used was the phonograph, with a series of special records to determine such factors as the appreciation of pitch, intensity, interval and consonance.

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3. Bryant, W. S.: A Phonographic Acoumeter, Arch. Otol. **33**:438, 1904.

4. Seashore, C. E.: University of Iowa Studies in Psychology, no. 36, Iowa City, Iowa University Press, 1920, vol. 1 and 2.

In following phonographic audiometry to its present day status, one notes as the next development after the Bristol machine the so-called 4-A audiometer of the Western Electric Company. This machine is a spring-wound phonograph with a magnetic pick-up.[5] The intensity of the speech (spoken numbers were used) is attenuated in the manufacture of the record. The sound is put out either to one receiver or to a group of twenty or forty receivers, so that classes of school children can be tested. The advantages (and disadvantages) of testing school children in groups were immediately taken up by the American Society for the Hard of Hearing, the members of which were anxious that deafness be detected early. This work is being followed with increasing popularity and service as the years go on.

Improvements are rapidly taking place in the acoustic field, and testing for the hearing of speech has been advanced by such apparatus as the motor-driven turntable, the audion bulb for amplification, the decibel meter and the crystal pick-up and crystal receiver. In addition to better mechanical equipment, there is a more thorough and scientific knowledge of speech and of speech hearing from pedagogic, psychologic and acoustic angles. Much careful research has been done by workers connected with the telephone industry in what they term their "articulation tests" (Crandall, 1917; Fletcher and Steinberg, 1929[6]). Otologists might preferably call these "intelligibility tests."

AUDIOMETRY OF THE RANGE OF PITCH[7]

In 1919 S. W. Dean, head of the department of otolaryngology at Iowa University, wrote:[8]

> . . . two years ago I decided that the methods of testing the tonal ranges used in our clinic must be improved upon. When I broached the matter to Professor Seashore he suggested the appointment of a research assistant to work in otology and psychology, and to solve the problem if possible.

C. C. Bunch was appointed to fill that position, and from this time dates the development of the scientific work in testing hearing across the range of frequencies.

Dr. Bunch's instrument passed through an evolution from the mechanical siren to an electrical siren dependent on the interruption of

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5. Macfarlan, D.: The Voice Test of Hearing, Arch. Otolaryng. **5**:1 (Jan.) 1927.

6. Crandall, I. B.: Composition of Speech, Physical Rev. **10**: 74 (July) 1917. Fletcher, H., and Steinberg, J. C.: Articulation Testing Methods, Bell System Tech. J., October 1929, p. 1.

7. Because the status of audiometric testing for bone conduction is indefinite, the history of the subject is not included in this article.

8. Dean, S. W., and Bunch, C. C.: The Use of the Pitch Range Audiometer in Otology, Laryngoscope **29**:453 (Aug.) 1919.

magnetic impulses by a rotating armature. "Steps regulating intensity can be varied at will and kept constant. All the notes in the tonal range can be tested with the same steps. The notes are relatively pure." Except in the refinement of method and quality and in the establishment of a unit of intensity, audiometry of the range of pitch has not advanced beyond the initial step made by Dean and Bunch. A large series of patients was tested; audiometer charts were made, and classification of types was attempted.

In 1921 Dr. John Guttman, of New York, read a paper before the Section of Otology of the New York Academy of Medicine in which he described an electric acoumeter.[9] I believe that this instrument was the first to use vacuum tubes for testing hearing. Intensity was controlled by changing the relation of primary and secondary induction coils. In 1922 Guttman published his audiograms secured in studies of different types of deafness.

About this period intense popular interest in radio was developing, and with it came a greater desire to know something of acoustics. Oscillators, such as the string galvanometer and Dayton C. Miller's phonodeik,[10] were used to record or to reproduce vibrations, while the generation of frequencies had been accomplished by electrically activating the fork. Beat tones, made by the interference of two pitches, had been known since Tyndall's time (1867), but with the appearance of the audion tube they were used to develop the beat frequency oscillator. Probably the most popular of these oscillators was that made by the General Radio Corporation of Cambridge, Mass.[11] It was to be found in most of the engineering schools and was at times used as a device for testing hearing. In a few years a considerable number of oscillating circuits were invented and established themselves.

The first frequency-run audiometer to be put out definitely for testing hearing was the Bell Laboratories' eight octave audiometer (known as the 2-A). Dr. Fowler was the otologic consultant during the development of this machine and began his many able contributions to the science of testing. Harvey Fletcher introduced this audiometer and published his classic article on the acoustics of various existing methods of test-

9. Guttman, J.: Further Studies in Functional Examination of Acuity of Hearing and Its Relation to Perception of Sounds of Different Pitches Produced by the New Electric Acumeter, Laryngoscope **32**:829 (Nov.) 1922. Tyndall, J.: A Course of Eight Lectures Delivered at the Royal Institution of Great Britain, New York, D. Appleton and Company, 1867.

10. Miller, D. C.: The Science of Musical Sounds, ed. 2, New York, The Macmillan Company, 1922.

11. General Radio Corp., Cambridge, Mass., beat-tone oscillator and electrically driven fork.

ing.[12] He introduced otologists to the new unit of intensity, the decibel, evolved from the "telephone," "transmission" or "sensation unit." The attenuator of the 2-A audiometer used the decibel meter.

In recent years, under the stimulus of the relative simplicity of oscillating circuits and the cheapening of radio equipment, many new audiometers have appeared. The cost of these machines has come down within the reach of many who could not previously afford them. In listing the following audiometers, I am setting down those which are at this date actively on the market, and I have omitted those that have not survived and those specially designed or custom made which some otologists are using.[13] To the list I have added oscillators adaptable for use in testing hearing.[14]

Western Electric Audiometer (Graybar)
Sonatone
Maico
Pilling
Brenco

The following oscillators are adaptable for testing hearing:

General Radio
R. C. A.
Clough-Brengle

(Numerous phonographs with electric pick-ups are now on the market; those using the crystal give faithful reproductions; all may be used for testing the hearing of speech in connection with a decibel meter and a proper record. The Graybar 4-A, supplanted by the 4-B, is the original type of this instrument and is available. Its range, however, does not extend beyond the measurement of a 30 decibel loss of hearing.)

STANDARDIZATION

Much progress has been made in an unostentatious way toward standardization of audiometers. Not only has the small group of interested otologists been attentive to the problems involved, but the cooperation of the manufacturers has been honest and sincere. The committee of the American Standards Association includes manufacturers, otologists and physicists. Its results are being coordinated with

12. Fletcher, H.: New Methods and Apparatus for Testing the Acuity of Hearing, Bell System Tech. J., September 1925, p. 1.

13. Jones, I. H., and Knudsen, V. O.: Audion Bulb Circuits in Otologic Practice, Arch. Otolaryng. **2**:335 (Oct.) 1925.

14. I have a considerable bibliography on the attempts at establishing pitch range audiometry in Europe. None of the instruments, however, reached a stage of perfection or of accuracy which made them generally acceptable. This bibliography is intentionally omitted.

those of the consultants on audiometers and hearing of the Council on Physical Therapy of the American Medical Association, for fortunately many of the same otologists are members of both committees.

It seems desirable, since this article is a history, to list the names of most of the American otologists and others who have made contributions to the development of audiometry: Dean, Bunch, Fletcher, Fowler, Seashore, Dayton C. Miller, Kranz, Pohlman, Sabine, Newhart, Guttman, Hayden, Knudsen, Witting, Isaac H. Jones, Guild, Sohier-Bryant, Edison, Bristol, Wegel, Crowe, Hughson, Lurie, Derbyshire, Davis, Polvogt, Steinberg, Goldstein, Stevens, Goodfellow.

One can be assured that great progress has been made in the measurement of hearing, especially when one recalls the erroneous methods and the unintelligent technic of the past. For the future, much improvement can be expected in the reliability of the various instruments and in their identity with one another so that results from various makes will be comparable.

1805 Chestnut Street.

DEVIATION OF THE SEPTUM IN RELATION TO THE TWISTED NOSE

SAMUEL SALINGER, M.D.
CHICAGO

I have often wondered whether my experience in surgical treatment of the nasal septum has been unique or whether in fact it parallels that of other rhinologists. It seems that my ideas on the subject have passed through several stages, the like of which I have noted in the experiences of many of the younger men under my observation, which are so distinct and vivid as to suggest that they are worthy of analysis for the sake of the lessons they may teach. When I was taking my postgraduate work in Vienna in 1914, having up to then had no actual experience in rhinologic surgery, I was led to believe that the operation on the septum was no. 2 on the list of surgical procedures to be learned, because of the frequency with which it is performed and also because it was supposed to be the easiest to master next to tonsillectomy. I had seen a great deal of this operation at the hands of Otto Freer, who had been my mentor and who at that time was internationally known for his work in perfecting the procedure, and I had acquired some rather definite ideas as to how it should be done. The principal points emphasized by Freer were proper exposure by means of his reversed L-shaped flap, dissection by sharp instruments, thorough removal of the vomer by means of chisels and his modification of the Greenwald forceps and above all thorough hemostasis and perfect illumination. Imagine my surprise, then, to find that few physicians in America or abroad were following his technic. Ballenger's swivel knife (which Freer decried) and the old Killian incision seemed to simplify the operation for most rhinologists, and the Kofler septum forceps for breaking and removing the bone were more widely employed than the more laborious and time-consuming biting with the Freer-Greenwald forceps. Having been trained in both methods, I also found the Killian-Ballenger technic simpler than the Freer and soon adopted it. It seemed to me at that time, after my first few experiences, that the operation was easy, and I felt pleased with myself. I discovered later that many features of the Freer technic were admirably suited to special conditions and worth adopting.

Read at a meeting of the Wisconsin State Medical Society, Milwaukee, Sept. 14, 1938.

In almost twenty-five years of experience as a teacher, I have observed this same cocksure attitude on the part of interns, residents and young practitioners toward the submucous resection, an attitude which I am sure was due for considerable revision. One is at first apt to excuse the perforations which occur early in one's surgical experience and to forget them in the contemplation of one's successful operations. The time it takes the rhinologist to realize that his early appraisal of his technical mastery of the submucous resection needs serious modification is no doubt variable, but I dare say that every honest surgeon sooner or later comes to the realization that the operation is at times far more difficult and complicated than he has ever realized and begins really to study his cases, especially his failures. He begins to wonder why in one particular case the membrane was badly torn and why in another instance the obstruction was not entirely removed. Gradually he begins to appreciate the fact that septums are not all alike and that one method of approach does not meet the requirements in every case. He begins to vary his technic and learns by experience how to overcome the difficulties which were previously overlooked or at best badly handled. So, frequently, the surgeon may be in practice several years before he is really satisfied with the sort of results he is getting. At this point he may find that, although he is doing a uniformly satisfactory resection of the septum, he occasionally meets peculiar situations that the textbooks fail to mention, and only by following the literature does he discover that these situations are really difficult and must be mastered by special methods.

One of them is the deviated septum whose lower free border has been dislocated into one nostril, a condition in which, as he knows from experience, disaster may follow total removal of the obstructing cartilage. Metzenbaum [1] has given this particular phase of surgical intervention on the septum much consideration and has done much to clarify the situation by evolving technical details and pointing out a method whereby the cartilage may be replaced without endangering the integrity of the nasal support. A second situation, which has received less attention, although some excellent articles have appeared in the literature, is that in which a fracture has so deformed the septum in a child as to offer a serious obstruction to his breathing. Here the surgeon is confronted with a condition which demands correction, and yet he is restrained from action by the age-old doctrine that it is not safe to operate on a child's septum because of the danger to the future development of the nose. I can heartily agree with this point of view but only so far as it

1. Metzenbaum, M.: Replacement of the Lower End of the Dislocated Septal Cartilage Versus Submucous Resection of the Dislocated End of the Septal Cartilage, Arch. Otolaryng. **9**:282 (March) 1929.

pertains to the classic submucous resection. This statement, however, does not imply that other methods are not available. Since this particular phase of the problem of the septum is not the direct object of my paper, I shall not dwell on it long. I should like merely to point out that a child's septum can be straightened and the airway restored without sacrificing any of the supporting structures and that I have done the operation repeatedly with good results. The operation is done with the patient under general anesthesia, supplemented by the injection of procaine hydrochloride and epinephrine for hemostasis, and consists of a submucous refracture of the obstructing bone and cartilage, with removal of an occasional narrow strip of the latter when overlapping is to be prevented, and the hacking of the cartilage to eliminate bends. The latter will be dealt with more in detail later in the article. Also the postoperative care by means of plugs of dental wax, which are sewed into place, will be fully explained since the same procedure is occasionally employed for adults.

The third type of septum which presents peculiar difficulties, and the one which will be the special topic of my remarks, is the deviated septum which is associated with an external nasal deformity. Most frequently this condition is the result of an old, untreated nasal fracture, and one is usually confronted with a twisted nose whose tip is off the median line and whose dorsum either is broad and depressed or has a bony protuberance that is likewise deviated from the midline. The problem here is to restore the airway and at the same time straighten the nose proper. In other words, one must envision a procedure which will restore functional utility and accomplish esthetic correction. At the present time there is still some difference of opinion as to whether this should be done in one sitting or divided into two separate operations, one for correction of the septum and subsequently another for straightening the nose or whatever cosmetic procedure may be indicated. In the majority of cases I am much in favor of the combined operation and have practiced it for several years with satisfactory results. I advocate it because, first, the patient is spared the double discomfort and expense of two operations; second, when intervention on the septum is to be followed immediately by a plastic operation on the nasal bridge, one is more likely to be conservative and therefore will remove no more cartilage and bone than is absolutely necessary, thereby avoiding the risk of weakening the nasal support, and, third, because the two operations may be complementary to each other in that when all the tissues are properly exposed one gains a better idea of how much or how little need be done to accomplish the dual purpose. I have seen a number of cases in which a preliminary submucous resection, done without consideration of the external deformity, so weakened the nasal bridge that it was extremely hazardous to attempt removal of a hump, shortening of

the nose or even straightening of a twist. On the other hand, if one were to attempt to straighten a crooked nose in the presence of a sharply twisted septum, expecting to resect the latter at a subsequent date, one would find that the septal deflection had a tendency to pull the nasal bones back to their abnormal position, thereby defeating the object of the operation.

Naturally there is no one technic that is applicable in all cases, since there is considerable variation in the types of deviation encountered, as well as in the external conditions which need correction. For practical purposes, therefore, it would be well to catalogue the types of deformity usually encountered and to consider the means by which in each instance the correction may best be accomplished. Before going into detail, I should like to point out three important items, applicable in all cases, which I have found extremely valuable in working out the special procedures required.

First, for anesthesia I use little cocaine, relying almost entirely on the injection of procaine hydrochloride, 1 per cent with epinephrine. By means of a long fine gage needle, beginning as far back on the septum as possible and working forward, one may thoroughly infiltrate the mucoperichondrium and obtain not only perfect anesthesia but adequate hemostasis, which is essential to satisfactory vision. One cannot do justice to the parts involved if the field is obscured by blood, and no amount of cocaine and epinephrine topically applied can compare with the hemostasis induced by the injection of procaine hydrochloride and epinephrine into the tissues overlying the bone and cartilage. Second, the septal cartilage may be moved from one position to another, sliced, partially excised and otherwise manipulated without danger to its vitality as long as it remains attached to its mucoperichondrium on one side. This point I feel has not hitherto been sufficiently stressed. It is therefore important when one wishes to conserve cartilage that the dissection be carried out fully on only one side. The cartilage may be cut loose from its dorsal attachment, from its attachment to the vomer and from the perpendicular plate posteriorly and safely replaced, even though its mucoperichondrium has been elevated on one side, as long as it still remains attached to the soft tissues on the other side. Third, cartilage that is curved may be straightened by hacking or by excision of narrow strips without impairing the vitality of the remaining portions. Sheehan [2] was one of the first to lay particular emphasis on this fact. Naturally one must avoid damaging or perforating the mucoperichondrium on the attached side. This, however, is easy, provided the field is clean, the illumination is good and a sharp elevator is applied on the bias.

2. Sheehan, J. E.: Plastic Surgery of the Nose, ed. 2, New York, Paul B. Hoeber, Inc., 1936.

OPERATIVE TECHNIC

As to special types, if the nose is twisted as a result of an old fracture and the septum is deviated, the procedure to be followed depends on whether one contemplates the removal of a hump or the shortening of the nose. In the absence of these two conditions and of dislocation of the free border of the septum, one is free to do the usual submucous resection and to follow it with refracture and reposition of the nasal bones. The technic of the latter has often been described and will here be only briefly sketched. The accompanying illustrations may help elucidate it. The incisions are made low in the nasal vestibule and carried directly to the margin of the piriform aperture. A small periosteal elevator is then introduced, which strips the periosteum from the bone along a line extending upward and inward toward the root of the nose, care being exercised to keep the intervention medial to the region of the lacrimal sac. With the elevator in place as a guide, a small Joseph saw is introduced into the pocket thus formed; the elevator is then removed and the bone sawed through. In performing this act, one should at first bear more heavily on the distal end of the saw, so as to be sure the upper or thicker portion of the nasal bone is cut through, and then transfer the weight of the cutting process to the lower end of the saw, which is going through the most medial portion of the ascending process of the superior maxilla. One can generally tell when the saw is approaching the mucous membrane on the inner surface of the bone, and it is well to desist at this point, lest the saw cut through this soft tissue, whose attachment to the bone insures its blood supply pending the reattachment of the detached periosteum on the outer side. When this procedure has been carried out on both sides, one is ready to infract the bones, provided the deviation does not extend up to the root of the nose. In many cases it is necessary to mobilize the nasal bridge completely by driving a sharp chisel through the root of the nose below the glabella. The chisel may be pushed through the skin, and when it is firmly applied to the bone it is not difficult to force it through by several sharp blows with a mallet. In order to be sure that the nose will fracture properly it is well to leave the chisel within the small incision it has created, and by shifting the loose skin to one side and then the other one may drive the chisel through on both sides without enlarging the incision in the skin. In this manner the entire nasal bridge has been severed so that it may be shifted by pressure with the fingers. One additional point must here be emphasized. There are certain cases in which the deviation of the nose has resulted in one side being flatter or broader than the other. To put it another way, the side opposite the deviation is often twice as broad as the other. In such a case the procedure just described will be inadequate, because when the nose is pushed

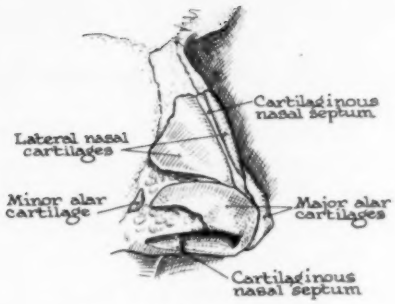

Fig. 1.—Cartilages of the nose.

Fig. 2.—Incision in the vestibule opposite the margin of the piriform aperture, first step in the procedure for straightening a deviated nasal bridge.

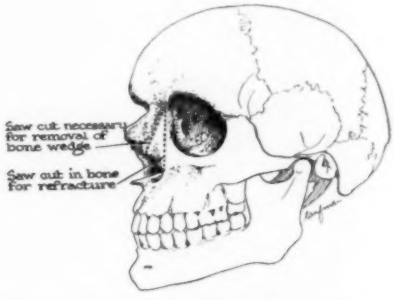

Fig. 3.—Saw cuts necessary for refracture of the nasal bridge.

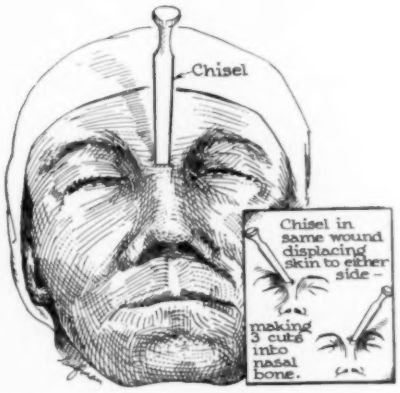

Fig. 4.—Procedure for severing the root of the nose with a chisel through a single incision the width of the chisel.

over to the flat side there will be overriding of the severed margins to such an extent as to predispose to a recurrence of the deformity. This is one of the most common causes of failure. It is therefore necessary to remove a segment of bone from the broad side of the nose by means of a second saw cut, whose upper end meets the first cut on that side but whose lower end (at the piriform aperture) is removed from the first cut by a distance varying from ⅛ to ¼ inch (0.318 to 0.64 cm.),

Fig. 5.—Copper molded splint and wax plugs in place.

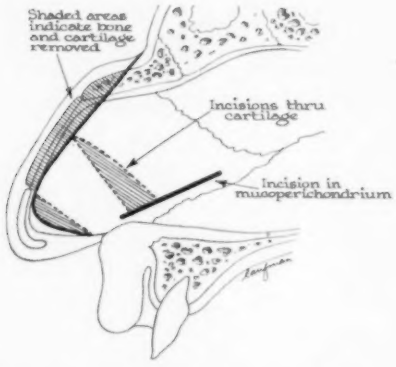

Fig. 6.—Combined procedure for removing a cartilaginous deviation, correcting a nasal hump and shortening the nose. The diagonal incision along the vomer is helpful in the removal of a low bony ridge.

according to the size of the wedge it is necessary to remove. This wedge is easily fractured by pressure with the periosteotome introduced into the pocket and removed by being peeled off the underlying mucosa. The nose is then packed and a copper molded splint applied to hold it in place. At the end of forty-eight hours the dressings are removed and the packing taken out, and after the nose has been cleaned the splint is

reapplied. Subsequently the splint is changed every forty-eight hours up to ten or twelve days, or until it is evident that the fragments are beginning to unite. In some cases I find the use of rolled dental wax superior to packing and have been able to leave it in the nose up to one week without danger. The advantage of the wax is that it does not entirely block the passages, secretions may escape around it and one avoids the disagreeable vacuum often produced in the nasopharynx by nasal packing.

Should it be necessary to remove a hump from the nasal bridge, it would be advisable to do this part of the operation first, because one would then be in a better position to judge how much of the septal cartilage and bone might be safely removed than if the procedure were reversed. In such cases I remove the hump by means of a Joseph saw and scissors and then do the submucous resection. Finally the nasal bones are infracted as described.

In cases in which, in addition to the conditions described, there is also a dislocation of the free border of the septum, it will be necessary to combine the procedures just outlined either with Metzenbaum's technic as he described it or with certain modifications to fit the individual case. The important thing to remember here is that one is attempting to preserve all the septal cartilage lying anterior to the angle of deviation. The incision is made along the free border of the septum and the mucoperichrondrium dissected off in the direction of the opposite nostril, up to the angle of deviation. At this point the cartilage is cut through along the angle of deviation, which frees all the cartilage anteriorly, so that it may be displaced with the blade of the speculum. (It should be remembered that this portion must be left attached to the mucoperichondrium on the side opposite the angle of deviation.) This procedure exposes the angle of deviation, and from here the dissection may be carried backward and upward on both sides, since this portion of the septum will have to be removed at least in part. If the portion of cartilage remaining is curved, it may be straightened by several incisions running parallel to the direction of the curve. In some instances I have found it unnecessary to remove more than a narrow, triangular strip of cartilage at the angle of deflection, or just enough to avoid overlapping when the anterior portion is brought back to the midline. In some cases it will be seen that the cartilage has been dislocated from the vomer and lies parallel to it and separated from it by both periosteum and perichondrium. In these cases it is impossible to replace the cartilage in its original groove, since the latter has filled with scar tissue and, furthermore, the cartilage is too wide from dorsum to nasal floor to permit such a maneuver. The cartilage cannot be swung back to the midline, because the bone is in the way. The remedy for this condition is to dissect the cartilage away

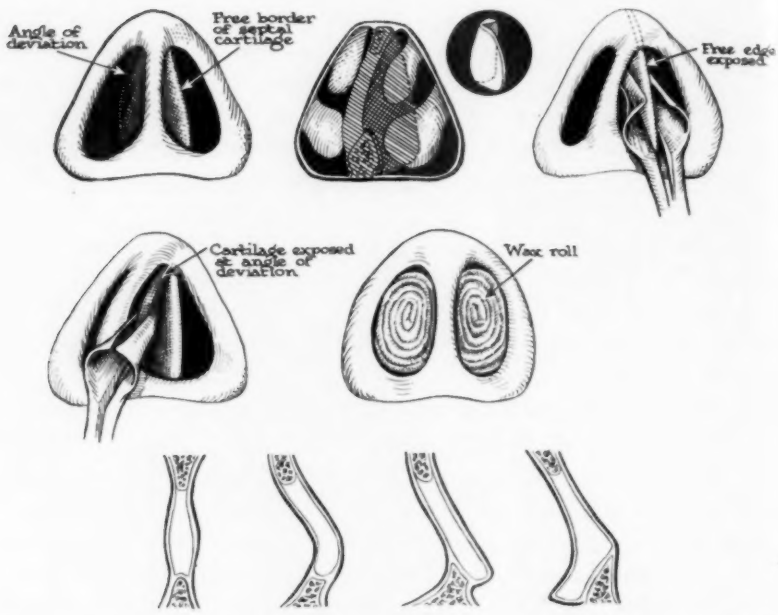

Fig. 7.—Top row, dislocated free border of the septal cartilage; deviation of the septum from before backward; exposure of the free border as the first step in the operative procedure. Middle row, septal cartilage freed back to the angle of deviation, where it is cut through and displaced temporarily to permit resection of the deviation behind; wax rolls in place. Bottom row, straight septum; curve involving only the cartilage; cartilage dislocated from the vomer and jutting into the opposite side; cartilage dislocated, showing angles in both sides.

Fig. 8.—Twisted nose; dislocated septal border.

from the bone and then chisel away enough of the latter to allow the cartilage room for its reposition in the midline. Its free border can then be readily inserted in the pocket previously prepared in the membranous septum, as described by Metzenbaum. If the cartilage is curved along its dorsal aspect, as shown in the accompanying illustration, it will be necessary to carry the dissection of the mucoperichondrium up to the skin of the dorsum and over the top of the cartilage. This procedure allows plenty of room for the cartilage after it has been straightened by hacking. Occasionally the nasal spine is displaced along with the cartilage and can be both seen and felt in the floor of the nostril.

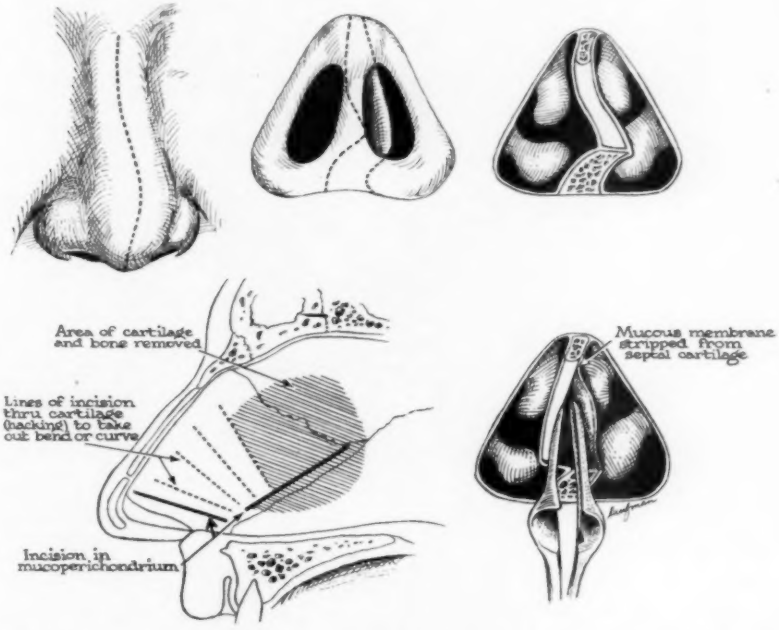
Area of cartilage and bone removed
Lines of incision thru cartilage (hacking) to take out bend or curve
Incision in mucoperichondrium
Mucous membrane stripped from septal cartilage

Fig. 9.—Top row, nasal twist involving the bony bridge and the septum. Bottom row, area of septum removed posteriorly while the curved anterior portion is straightened by hacking; curved anterior portion held aside by the speculum after being detached from the vomer to permit resection of the obstructing bone.

The remedy in this case is to loosen it with a chisel and push it back to the median line along with its superimposed cartilage, which can be done by pressure with the thumb. In many of the cases in which the free border of the cartilage is repositioned with or without the nasal spine, I find a tendency to recurrence unless some measures are taken to hold the nose in place for a sufficient time. To accomplish this, I use two rolls of dental wax, which are held in place by a through and

through mattress suture including both rolls and the septum. These wax plugs cause absolutely no irritation to the mucous membrane and are readily borne for a week. For children, when it is necessary to refracture a badly deviated septum, this method of dressing has proved unexcelled, and I can recommend it most highly. The wax is nonirritating; secretions pass it readily, and it softens sufficiently under the warmth of the tissues to accommodate itself to the shape of the intranasal structures.

Again, there are cases in which it is advisable to shorten the nose in addition to the other procedures outlined. In such cases one performs all the other maneuvers indicated, being particularly careful to leave sufficient cartilage below and anteriorly so that the final step, namely, removal of a strip from the lower free border, will still leave plenty of cartilage to support the nose. As is shown in the illustrations, it may require two incisions in the septal mucosa separated by an area attached to the cartilage, which is left in place. The only cases in which the

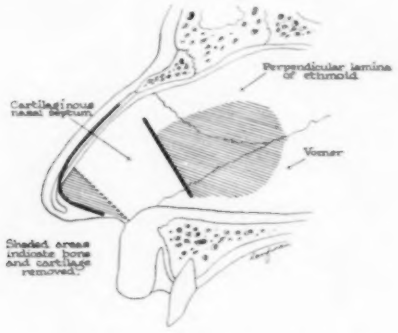

Fig. 10.—Incisions necessary for combined resection of the septum and procedure for shortening a long nose.

operation may be done through a single incision are those in which the free border of the septum has been displaced.

Finally I shall say a few words about postoperative treatment. As has been stated, the initial dressings are left in place for forty-eight hours unless symptoms of local infection should appear. Since my experience is limited principally to the use of the copper molded splint, as I have long since given up the head band apparatus with external pressure pads except in treating recent fractures which involve other facial bones, I can speak for only this one method of treatment. The copper molded splint causes no pain. If the patient complains of pain in the nose, particularly localized pain, and if in addition the edema which usually follows a refracture becomes red and fever is present, one may suspect an infection. The indication here is promptly to remove all dressings and packing and at once order hot wet dressings of

magnesium sulfate. Frequently suppuration will be avoided. However, occasionally there will be signs of fluctuation. It is then necessary to make an external incision for drainage. This will undoubtedly leave

Fig. 11.—Nasal twist due to an old fracture of the septum.

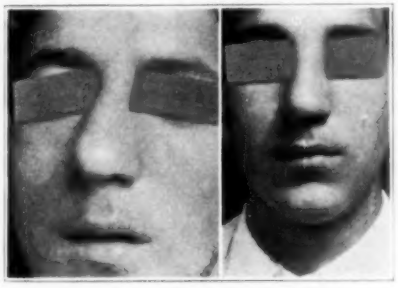

Fig. 12.—Combined deviation of the bony and the cartilaginous bridge.

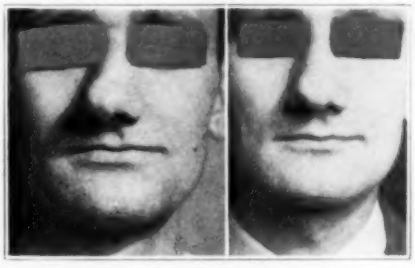

Fig. 13.—Extreme deviation of the cartilaginous portion of the nose due to an old fracture. The septum is markedly twisted.

a scar, which, however, may later be excised and considerably minimized. Fortunately infections are rare. When they do occur, they may be attributed either to a recent, uncured infection of the upper part of the

respiratory tract or to a prevalent epidemic of influenza. It is therefore extremely important to avoid operating too soon after a cold, in the presence of suppurating sinusitis or during an epidemic of severe colds. Moreover, during the postoperative period the patient should avoid contact with persons suffering from acute infections of the respiratory tract. I have seen several infections develop four to seven days after operation, which I was convinced were due to exposure.

The treatment of postoperative edema of the lids consists of cold applications maintained for twenty-four to forty-eight hours by means of small pads of cotton saturated in ice water or a weak solution of aluminum subacetate.

If packing is used, it should be removed in forty-eight hours and not replaced. If wax plugs are used, they should be left in place for five to seven days. The copper splint is changed every forty-eight hours and adjusted to meet the conditions. I feel that much of the prolonged postoperative thickening encountered with other methods of treatment has been avoided by this procedure, since it insures firm and steady application of the soft tissues to the underlying bone and prevents stasis and edema. Sutures are employed only in replacing a dislocated septal cartilage or in the procedure for shortening a nose. In the former case they involve only the mucoperichondrium and may be removed in forty-eight hours. In the latter case they are passed through the septum and columella and should be left undisturbed for five to seven days.

25 East Washington Street.

Case Reports

THROMBOPHLEBITIS OF THE LATERAL SINUSES COMPLICATING BILATERAL ACUTE MASTOIDITIS

MORLEY THOMAS SMITH, M.D., NEW ROCHELLE, N. Y.

One of the most difficult problems that face the otologist, from a standpoint of both diagnosis and of treatment, is that presented by the case in which signs of sepsis occur after a double mastoid operation has been performed. It is difficult because even after all the means at one's command, including inspection of the sinuses, have been used, it is often impossible to tell with any degree of certainty which sinus may be the offender. If after one jugular vein has been ligated and the sinus obliterated the symptoms still persist, the problem of proper procedure is further complicated.

That bilateral thrombophlebitis with or without complete thrombosis, diagnosed ante mortem, is not a common condition is attested by the facts that few cases are on record and that many competent observers with wide experience, working in large centers, have not encountered the condition.

REVIEW OF THE LITERATURE

A search of the literature to date reveals that 3 cases of bilateral thrombophlebitis of otitic origin with recovery following ligation of both internal jugular veins and obliteration of the sinuses have been reported: the cases of Hastings,[1] reported in 1926; Ruttin,[2] in 1925, and Despons,[3] in 1932. There have been 6 cases of bilateral thrombophlebitis of otitic origin with recovery following ligation of one internal jugular vein and obliteration of both lateral sinuses. These cases were reported by

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1. Hastings, H.: Bilateral Jugular Resection for Bilateral Sigmoid Thrombosis (Otitic), Arch. Otolaryng. **4**:58 (July) 1926.

2. Ruttin, E.: Ueber doppelseitige Jugularisunterbindung und Schädigungen durch Jugularisunterbindung überhaupt, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **22**:156, 1925.

3. Despons, J.: Bilateral Phlebitis of the Lateral Sinus with Bilateral Ligation of the Internal Jugular, Rev. de laryng. **53**:521, 1932.

Gruening,[4] Smith,[5] Stickney,[6] Tobey,[7] Dausend[8] and Schlegel.[9] My case falls in the latter category.

Hastings, in his review of the literature in 1926, found 5 cases reported by general surgeons in which both jugular veins had been ligated during operations for cancerous or tuberculous conditions in the neck; in none of them, apparently, was the condition of otitic origin. There was 1 operative death in this group of 5 cases.

Hastings' interesting case was described in detail, and many salient points in the diagnosis and treatment of this condition were brought out. In his case, the operation on the sinuses and the jugular veins were done seventeen days apart. It is of interest to note that at the time of exploration and operation on the right sinus and jugular vein the left mastoid appeared firmly healed, the hearing was normal and there was no reason to suspect trouble on that side. The author stated that there were swelling and tenderness over the upper part of the right side of the neck in the region of the internal jugular vein and that up to this time he had suspected abscess of the brain rather than thrombophlebitis. The ophthalmologic findings of hazy disks and engorged veins antedated the operative work on the sinuses and the ligation of the veins. He commented on the fact that the left vein was thrombosed at operation, as the right one had been, and that obstruction to the circulation on this side had been going on for some time before the second operation.

In Ruttin's case seven weeks intervened between the operation on the sinuses and that on the jugular veins. Ruttin cited untoward events that have followed ligation of one internal jugular vein, such as choked disks and, occasionally, death. He explained them as being due to the incapacity of the sinojugular venous system on the opposite side to accommodate the sudden increase in the flow of blood in the cranial cavity and advised that an interval of time be allowed to elapse between operations.

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4. Gruening, E. P.: A Case of Bilateral Thrombosis of the Sigmoid Sinus; Recovery, Tr. Am. Otol. Soc. **10**:518, 1907.

5. Smith, C. H.: Double Mastoid with Septic Sinus Thrombosis, New York M. J. **101**:407, 1915.

6. Stickney, O. D.: Bilateral Acute Suppurative Otitis Media with Symptoms of Sinus Thrombosis, Laryngoscope **29**:90, 1919.

7. Tobey, H. G.: Double Acute Suppurative Mastoiditis with Symptoms of Sinus Phlebitis or Thrombosis, Tr. Am. Laryng., Rhin. & Otol. Soc. **29**:6, 1923.

8. Dausend, J. A.: Ein Fall von doppelseitiger Sinusthrombose im Anschluss an einseitige Otitis media purulenta mit Ausgang in Heilung, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **4**:394, 1910-1911.

9. Schlegel, G.: Ein Fall von doppelseitiger Sinusthrombose mit einseitiger Jugularisunterbindung, Arch. f. Ohrenh. **69**:176, 1906.

Kopetzky [10] made a practical observation in commenting on the cases of Hastings and Ruttin. He stated that only in the rarest instances is there likely to be an equal and simultaneous growth of a thrombus in each sinus, that the result of the unequal growth is earlier obstruction on one side than on the other and that this factor permits gradual adjustment of the collateral circulation. He too brought out the point that an interval should elapse between the operations to allow the adjustment to be effected. He further commented that despite the large number of cases in which sinus thrombosis is seen at autopsy there are only rare instances of bilateral involvement.

Braun,[11] in his volume on thrombophlebitis of the sinuses, gave a complete anatomic picture of the venous channels of the head and neck and graphically showed the many variations and anomalies that may occur. He also fully described the collateral circulation that is established after one or both main venous channels have been blocked. It is obvious from a study of this work that various arrangements and anomalies may exist and that in a case of bilateral thrombophlebitis in which one lateral sinus happened to be small or absent, the prospect of recovery following bilateral operation on the sinuses and the jugular veins would be diminished.

Despons described the case of a boy 15 years old with bilateral otitis media, who recovered after bilateral mastoidectomy and bilateral operation on the sinuses and the jugular veins.

Alden [12] reported a case which he described as one of aseptic thrombosis of both lateral sinuses. It was his opinion that the condition arose slowly allowing the collateral circulation, which he concluded was by way of the inferior petrosal sinuses, to be established. The left sinus only was opened in this case. Sepsis did not occur, and the patient recovered. Alden stated that at no time during convalescence was there any swelling of the head or other evidence of block to the circulation from the brain. Examination of the eyegrounds was not mentioned.

McCoy [13] reported the case of a boy aged 9 years who had a simple mastoid operation on the left in June 1919 and three weeks later had an operation on the sinus and the jugular vein on this side. In February 1921 the right ear became involved, and simple mastoidectomy was subsequently done on that ear. Sepsis continued, and the blood stream was infected with hemolytic streptococci. On March 21 the right sinus was opened, and a mural clot was found, but there was free bleeding

10. Kopetzky, S. J.: Acute and Chronic Otitis Media and Sinus Thrombosis, Arch. Otolaryng. **6**:162 (Aug.) 1927.

11. Braun, A.: Sinusthrombophlebitis, New York, Paul B. Hoeber, 1928.

12. Alden, A. M.: Unusual Cases of Lateral Sinus Thrombosis, Ann. Otol., Rhin. & Laryng. **38**:459, 1929.

13. McCoy, J.: Sinus Thrombosis Following Mastoiditis, Tr. Am. Acad. Ophth. **26**:341, 1921.

from the lower end, and in view of this fact the surgeon decided not to ligate the internal jugular vein. The patient recovered. This case, of course, is in many respects not comparable to the cases under discussion, because of the length of time that elapsed between the two operations.

REPORT OF CASE

H. W., a white boy aged 10 years, was admitted to the hospital on Jan. 6, 1935, complaining of profuse discharge from both ears, some headache and elevation of temperature.

History.—Two weeks prior to admission the patient had an acute head cold and sore throat; two days after the onset of these symptoms, a rash was noted over his entire body, and the family physician made a diagnosis of scarlet fever. Coincident with the development of the rash, both ears began to discharge, but the patient did not complain of any pain. He had a fever during the interval, the highest temperature being 104 F. The rash disappeared within four days, and the symptoms in the throat cleared up. The temperature was still in the neighborhood of 102 to 103 F.; the ears continued to discharge profusely, and the patient's condition was one of apathy. Just before his admission to the hospital the temperature rose to 106 F. During this time there was marked impairment of hearing in both ears.

The patient's earlier history, including that of the ears, was essentially noncontributory.

Physical Examination.—On admission the patient appeared to be in a toxic condition; he was apathetic, the skin being dry and the tongue coated. Marked impairment of hearing was noted, but the patient answered questions correctly when aroused. The temperature on admission was 106 F., the pulse rate 120 and the respiratory rate 20. Neurologic examination, including inspection of the eyegrounds, showed no abnormality. Examination of the ears showed both canals filled with pus. There were sagging and narrowing of both fundi, the landmarks being entirely obliterated. There was no edema or tenderness over either mastoid. Tonsils and adenoids were present, but there was no evidence of infection in the nose or throat at the time. Examination of the chest showed a few scattered rales in the base of the left lung. A roentgenogram of the mastoids showed bilateral destructive acute mastoiditis.

A complete blood count on admission showed: leukocytes, 19,000; erythrocytes, 4,190,000; hemoglobin, 78 per cent; neutrophils, 90 per cent, and young forms, 17 per cent. The urine was normal.

Bilateral Simple Mastoidectomy, January 7.—The mastoids were large, pneumatic and extensively involved throughout, most of the cellular structure having been destroyed. The cells extended somewhat behind the sinuses; both mastoid tips were necrotic. Necrosis extended into the zygomatic areas. The deep row of cells anterior to the vertical portion of the lateral sinus and below the level of the facial nerve also was found to contain pus and granulations. There were no pathologic exposures of the sinuses or of the dura of the middle fossa. The sinus plate was removed on each side below the knee for a distance of approximately 1.5 cm. for the purpose of inspecting the condition of the lateral sinuses, both of which appeared grossly normal. A complete exenteration of both mastoids was done; one Penrose drain was carried up to each antrum, and the mastoid cavities were lightly packed with iodoform gauze. Operation was uneventful.

Material for culture was taken from both mastoids, and pure hemolytic streptococci were grown. A blood culture also was made at this time; there was no growth after seventy-two hours' incubation.

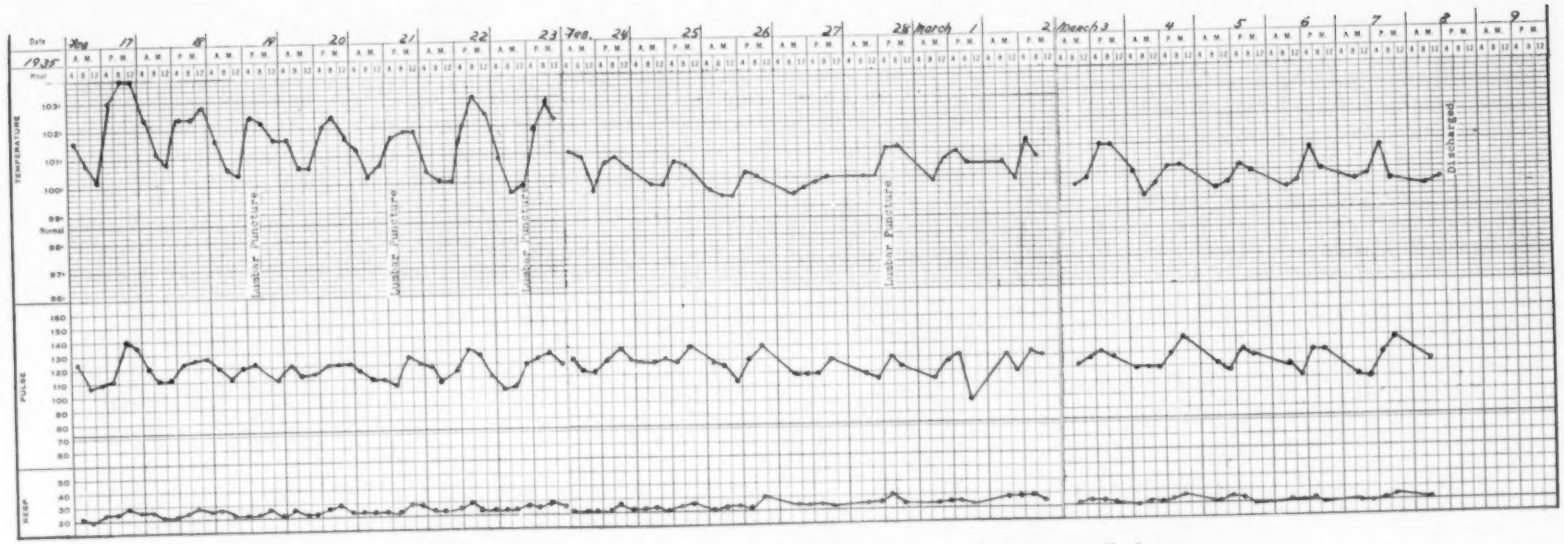
Postoperative Course: As will be noted from the chart, the temperature remained high with some remissions, but the patient's general condition showed some improvement. There was moderate drainage from both wounds, and the exposed sinuses did not show anything significant at this time. Two days after the operation the patient seemed slightly less drowsy, and the skin was moist. The temperature remained high, and, in spite of the negative blood culture, a tentative diagnosis of infection of the blood stream was made. On January 10 the patient was given a transfusion of 250 cc. of blood. It was thought advisable to delay investigation of the lateral sinuses for a few days, because not infrequently in this condition clinical signs of sepsis appear but after adequate surgical intervention in the mastoid region, plus transfusions, many of the patients make an uneventful recovery without further operative intervention.

The patient, however, complained of intermittent headache, was restless and drowsy and the day following the bilateral mastoidectomy had a chill which lasted ten minutes; he complained of some abdominal pain, and there was considerable abdominal distention as well as a troublesome cough. Examination of his chest at this time showed subcrepitant rales in the left lower part but no consolidation, and it was thought that this condition was contributing to his elevation of temperature.

A blood culture on January 14 showed a profuse growth of hemolytic streptococci. The patient was given another transfusion of 300 cc. of blood on this date, and at this time it was decided to explore the mastoids and the lateral sinuses. It is to be noted that on January 10 the patient complained of severe pain in the region of the right elbow.

Operation on the Right Sinus and Jugular Vein, January 15.—Little attempt at repair was noted in either mastoid cavity, the granulations being pale and unhealthy looking. The left lateral sinus, which had previously been exposed at operation, did not show anything grossly abnormal. Inspection of the right lateral sinus, however, showed it to be covered with pale, thickened granulations; it felt firm to pressure and gave the appearance of bulging; no pulsation in the sinus could be felt. This appearance of the sinus was evident from the extreme lower end backward a distance of 2.5 cm. beyond the knee of the sinus. At this stage it was decided to ligate the internal jugular vein, which was done below the level of the facial vein. There was no gross evidence of a pathologic condition in the jugular vein or in the carotid sheath. The vein was ligated with two chromic gut sutures and was not divided. The wound in the neck was closed with skin clips. The lateral sinus was uncovered until healthy-looking dura appeared in all directions, and on incision of the anterior wall of the sinus it was found to be blocked by a well organized clot, the wall of the vein appearing markedly thickened and gray. The clot extended from the extreme lower portion of the sinus backward to 3 cm. beyond the knee, at which point free bleeding was obtained. The center of the clot showed some signs of degeneration. There was no bleeding from the bulbar end of the sinus. The mastoid emissary vein was thrombosed, and there was slight bleeding from the superior petrosal sinus. The infected part of the anterior wall of the sinus was excised. Bleeding at the posterior end was controlled with plugs of iodoform gauze, as was the bleeding from the superior petrosal sinus. The patient was given a transfusion of 340 cc. of blood while on the operating table. His immediate postoperative condition was satisfactory.

Date
1935
Hour
TEMPERATURE
PULSE
RESP
Normal
Jan. 6
7
8
9
10
11
12
Jan. 13
14
15
16
17
18
19
Jan. 20
21
22
23
24
25
26
Bilateral Mastoidectomy Operation
Incision
Transfusion 450 cc.
Transfusion 300 cc.
Right sinus & jugular Operation
Hypodermoclysis - 500 cc saline in dextrose
Transfusion 210 cc. Blood
Lateral Sinus Operation
Transfusion 240 cc. Blood
Hypodermoclysis - 500 cc. Dextrose 10 saline
Jan 27
28
29
30
31
Feb. 1
2
Feb. 3
4
5
6
7
8
9
Feb. 10
11
12
13
14
15
16
Intravenous - 50 cc. - 50% dextrose
Lumbar Puncture
Transfusion 300 cc.
Incision + Drainage Right Knee
Lumbar Puncture



Graphic representation of the patient's condition from admission to discharge.

Postoperative Course: The patient's temperature continued to be of the septic type with wide variations, as will be seen by the chart. He was given another transfusion of 210 cc. of blood on January 18. He took fluids fairly well, remained drowsy and appeared to be in a toxic condition, showing considerable emaciation in comparison with his condition at the time of admission. Two days after operation, at 8 a. m., the temperature was 96.6 F., the pulse rate 56 and the respiratory rate 20; in the evening the temperature rose to 105.2 F., the pulse rate to 110 and the respiratory rate to 38. At this time the patient was slightly cyanosed and had some respiratory difficulty. It is to be noted that examination of his fundi up to this point had not shown any changes.

On January 20 the wound in the neck had healed per primam, and when the plug of iodoform gauze was removed from the end of the right sinus nearest the torcular Herophili, no bleeding occurred and the wounds appeared in satisfactory condition. The swelling and pain in the right elbow persisted, and there was considerable pain on extension of the forearm. Blood culture on January 21 still showed a profuse growth of hemolytic streptococci.

It was evident at this time from the clinical findings, together with the persistent positive blood culture that the entire infection had not been eliminated from the blood stream. A consultation was held with the general surgeon, the neurologist and the pediatrician, and the opinion was that the infection was in all probability coming from the venous sinuses. The boy's condition at this time was desperate, and it seemed that if this infection was to be controlled the only recourse left was to interrupt the circulation on the left side.

Left Lateral Sinus Operation on January 22.—The sinus, which had previously been exposed, was found to be covered with unhealthy-looking granulations, but the vein appeared flexible, showed pulsation and did not appear grossly to contain a clot. After the wall of the sinus had been sterilized, a hypodermic needle was inserted into it, and blood was readily obtained, there being no sensation of a clot when the vein was entered. It was decided not to tie the internal jugular vein at this stage but to block the sinus above the knee and at the lower end and excise the anterior wall of the vein. When this was done, free bleeding from both ends of the vein was found, and there was no gross evidence of clot. Free bleeding from the superior petrosal sinus also was noted. There being no evidence of a thrombus in the vein in the exposed area, the jugular vein was not tied, as it was felt that this omission would leave a by-pass to help take care of the cranial circulation by way of the inferior petrosal sinus. However, had there been a clot in the vein, the internal jugular vein would have been tied off at this time, or later if sepsis had not been controlled. Some surgeons feel that it is not necessary in all cases of this type to ligate the internal jugular vein. I have, on two or three occasions, because of the emergency at hand, omitted this procedure, and the patients have progressed satisfactorily, but I feel that in most cases and particularly in those in which only one side is involved, the internal jugular vein should be ligated at the time of the operation on the sinus. The patient's immediate postoperative condition was satisfactory.

Postoperative Course: The patient was nauseated and vomited at intervals for three days following the operation and had occasional nausea for one week thereafter. The signs of sepsis began to abate, as will be seen from the chart. Blood culture the day after operation was reported negative, and subsequent cultures, on January 30 and February 18, were negative. From the clinical course and bacteriologic reports, the septic process seemed to be controlled by January 25. Signs of increased intracranial pressure soon began to appear. Headache for the first few days was of moderate intensity and intermittent, but at the end of five

days was intense; the patient cried out frequently, was restless and vomited occasionally.

Neurologic examination on January 27 showed the patient to be well oriented, cooperative and drowsy. The deep reflexes showed slight accentuation on the right side, and there was generalized hyperesthesia with a suggestion of Kernig's sign on the right. The optic disks showed tortuous dilated veins, more marked on the left, with bilateral papilledema. Examination of the fundi after the first operation on the sinus and the jugular vein showed no abnormality. The neurologist advised against spinal taps at this time because it was not certain that the infection of the blood stream had been eliminated and there was a possibility of carrying infection into the meninges in doing a tap. On his recommendation the patient was given 50 cc. of 50 per cent solution of dextrose intravenously every eight hours for three doses, for the purpose of reducing intracranial pressure. The patient was given eight such doses of dextrose over a period of five days. On January 29, examination of the fundi showed an increase in the edema, there being between 2 and 3 diopters of swelling in each disk, with small hemorrhages below the right disk and on the nasal side of the left disk. Daily neurologic examinations were made, but no signs of intracranial involvement other than that accounted for by the vascular changes were ever noted.

On February 1 the first spinal tap was done. The fluid was clear and under a pressure of 460 mm. of water; 50 cc. of fluid was withdrawn at this time. Examination of the spinal fluid showed 4 cells per cubic millimeter, a small amount of albumin and globulin and normal reduction of Fehling's solution. Cultures of this specimen and of several subsequent ones did not reveal any growth or any essential change. Spinal tap was repeated from February 1 to February 28, eight times in all; at the last tap, on February 28, the pressure was 180 mm. of water. The fluid was always slowly withdrawn, from 15 to 20 cc. each time. The severe headache persisted until February 19, but after each spinal tap the patient had some relief for a few hours, and the decrease in the headache ran parallel to the decrease in the edema of the disks.

The patient was given five transfusions in all between January 10 and February 5. I feel that in all cases of this type blood transfusion is an invaluable therapeutic agent and should be used early in the disease and repeated as frequently as the condition warrants, small amounts being given at each transfusion.

Referring to the chart, it will be noted that the patient had considerable fever from February 11 to February 24. This fever in the beginning was thought to be due to a metastatic abscess in the right elbow, which was opened and drained by a general surgeon. Culture of the pus showed pure growth of the hemolytic streptococcus. One week later the infection in the elbow had subsided, and no further metastases or other signs could be found to explain the persistent elevation of temperature.

A complete blood count on February 13 showed: leukocytes, 9,500; erythrocytes, 3,930,000; hemoglobin, 68 per cent; polymorphonuclears, 64 per cent, and lymphocytes, 35 per cent. A count done on February 26 showed: leukocytes, 22,350; polymorphonuclears, 17 per cent; lymphocytes, 83 per cent, and young forms, 42 per cent. Tests for Lehndorf's heterophilic antibody reaction showed no agglutination of sheep's blood cells in any dilution, but a medical consultant considered the picture that of infectious mononucleosis, which he thought sufficient to explain the temperature. Examination of the upper part of the respiratory tract at this time showed no abnormality. The patient was given a preparation of liver and stomach extract and iron (lextron) and halibut liver oil and exposed to ultraviolet radiation. A blood count on March 7 showed: leukocytes, 18,050;

erythrocytes, 3,620,000; hemoglobin, 70 per cent; neutrophils, 15 per cent; lymphocytes, 84 per cent; central pallor, moderate; occasional normoblasts; no megaloblasts, and moderate polychromatophilia. Several blood counts have been made since the acute illness and have persistently shown the lymphocytes predominating. The last count, done a few months before the time of writing, showed: leukocytes, 4,050; erythrocytes, 3,980,000; hemoglobin, 76 per cent; polymorphonuclears, 28 per cent, and lymphocytes 70 per cent.

Toward the end of February the patient was happy and alert, slept and ate well and was out of bed for short intervals. On March 8 the wounds were healed; the middle ears were dry; the disks showed slight swelling of the margins, and the hemorrhages had entirely disappeared, vision being normal in each eye. The patient was discharged from the hospital on that date.

Since his discharge from the hospital the patient has been well, has shown normal growth, has taken an active part in school athletics and has had a satisfactory scholastic record. About three years has elapsed since his illness, seemingly sufficient time to bring forth any delayed neurologic or mental symptoms resulting from this severe sepsis and the surgical procedures.

COMMENT

As to the question of how the infection travels from one side to the other in such cases and whether there is a direct communication between the two lateral sinuses, there is considerable difference of opinion. Braun [11] stated that the thrombus may extend either with or against the blood stream but that it rarely passes to the opposite side. Page [14] quoted Richards [15] as saying that a thrombus cannot extend from the opposite side of the torcular Herophili because of the rapidity of the blood stream at the junction of the superior longitudinal sinus with the lateral sinus. Ballance [16] supported this theory in general, but stated that the rule is not invariable.

In my case it is open to question whether the infection extended from the right to the left lateral sinus or whether the left sinus was infected farther back on the same side, showing no gross changes in the area of sinus exposed at operation. After the blocking of the left sinus the course was stormy for several days, owing to the abrupt obstruction of the circulation. It will be recalled that there was complete thrombosis in the sigmoid, and in part of the transverse portion

14. Page, J. R.: Case of Septic Thrombosis of the Jugular Bulb with Repeated Formation of Septic Thrombi in the Sigmoid and Lateral Sinuses with Reference to the Literature on Involvement of the Torcular Herophili in Such Cases, Ann. Otol., Rhin. & Laryng. **25**:595, 1916.

15. Richards, J. D.: A Case of Infective, Lateral, Sigmoid, Superior Petrosal Sinus and Jugular Thrombosis; Operation; Recovery, Am. J. M. Sc. **129**:261, 1905.

16. Ballance, C. A., and Hobhouse, N.: A Case of Septic Thrombosis of the Left Sigmoid, Left Cavernous, and Left Inferior Petrosal Sinuses, with a Suggestion for Treatment in Future Cases, Ann. Otol., Rhin. & Laryng. **21**:942, 1912.

of the right sinus, whereas on the left side there was no demonstrable clot, which means that there had probably been no attempt to establish collateral circulation, such as occurs when gradual thrombosis is in progress. In several of the cases reported thrombosis had been going on for some time prior to operation, which accounts for the fact that the intracranial symptoms were apparently less marked than in the case reported here.

It is my opinion that this condition occurs more frequently than one would judge from the literature and that the small number of recoveries reported is due to the belief of otologists that fatality would ensue if both main venous channels were obliterated. These cases reported by both otologists and general surgeons seem to establish the fact that both lateral sinuses may be obliterated with ligation of one or both internal jugular veins, providing sufficient time has elapsed between operations to allow for establishment of collateral circulation. Eagleton [17] supported this procedure, stating that the anastomosis of the arterial supply is so nearly complete that surgical obstruction of any one set of large vessels will generally not seriously interfere with its function and that this applies in a much greater degree to the venous return, although the latter is occasionally upset disastrously by surgical intervention.

Patients with this condition usually present a desperate picture, and there is no doubt, judging from the recoveries reported, that many of them may be saved if the sinuses are blocked and the veins tied and that if this is not done the condition is likely to terminate fatally. The case here presented well illustrates this point.

SUMMARY

A review of the literature of cases of bilateral thrombophlebitis is presented.

A case of bilateral thrombophlebitis in which the blood stream was infected with hemolytic streptococcus is reported; the patient recovered after operation.

The postoperative treatment for the control of secondary hydrocephalus is outlined.

Metastasis in the right elbow was followed by mononucleosis due to infection.

Bilateral operation on the sinuses and the jugular veins may be the means of saving the lives of many of the patients.

Hemorrhages into the brain as a result of this procedure may cause serious complications, both acute and latent.

17. Eagleton, W. P.: Cavernous Sinus Thrombophlebitis and Allied Septic and Traumatic Lesions of the Basal Venous Sinuses: A Clinical Study of Blood Stream Infection, New York, The Macmillan Company, 1926.

CARCINOMA OF THE EPIPHARYNX

A Peculiar Case

HANS BRUNNER, M.D., CHICAGO

Carcinomas by contact are relatively rare in the upper airways and digestive passages. I agree with Wiethe's definition of tumors by contact as tumors which develop by implantation of tumorous elements on a surface constantly or frequently touching the tumor. These tumors by contact have to be distinguished from real tumors by inoculation, which develop by the patient's either swallowing or aspiring parts of primary tumors in higher situations or vomiting, expiring or coughing parts of those in lower situations. As far as real tumors by inoculation are concerned, a number of cases are known (collected by Wiethe [1]). Certainly some pathologists of great experience, such as Fischer-Wasels do not quite believe in the frequency of these tumors. But since this paper deals with tumors by contact and not with tumors by inoculation, this question is passed over.

St. Clair Thomson and Colledge [2] mentioned only the cases of Newman and Semon, in which carcinomas by contact occurred on both vocal cords. Particularly the case of Newman is instructive. The patient had first a carcinoma of one vocal cord. Where this carcinoma touched the cord of the healthy side a redness appeared. Later a carcinoma developed in the red place on the healthy vocal cord. However, Kahler [3] emphasized that in the region of the vocal cords a carcinoma by contact can be imitated, in that a carcinoma of the cord creeps along the subepithelial lymphatic space until it reaches the other side, where it breaks through.

As far as the upper parts of the digestive tract are concerned, Kahler has observed a few circumscribed tumors at the posterior surface of the cricoid plate in a case of carcinoma of the sinus piriformis. He considered the tumors at the cricoid plate carcinomas by contact. Vogelsang [4] has described a case (his case 2) of round cell sarcoma of the epipharynx and soft palate. He considered the findings to indicate multiple tumors of the upper digestive passages. I believe that the tumor of the soft palate was a tumor by contact, since I have seen similar conditions in the following case.

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1. Wiethe, C.: Beitrag zum Studium der metastatischen Larynxtumoren, Ztschr. f. Hals-, Nasen- u. Ohrenh. **21**:623, 1928.

2. Thomson, St. C., and Colledge, L.: Cancer of the Larynx, New York, The Macmillan Company, 1930.

3. Kahler, O.: Die bösartigen Neubildungen des Kehlkopfes, in Denker, A.: Handbuch der Hals-, Nasen- und Ohrenheilkunde, Berlin, Julius Springer, 1929, vol. 5, p. 408.

4. Vogelsang, W.: Ueber multiple Sarkome des Rachens, Ztschr. f. Ohrenh. **61**:330, 1910.

REPORT OF A CASE

K. S., aged 64, was treated with roentgen rays from May until December 1934 because of a glandular tumor on the left side of the neck (dose 9,250 r.). At that time no pathologic condition in the nose had been found. In May 1935 the treatment with roentgen rays was repeated (dose 1,400 r.). On June 14 Dr. E. G. Mayer undertook the treatment with roentgen rays. At the beginning of the treatment there was a humpbacked tumor in the epipharynx. The openings of the eustachian tubes could not be seen. A large, fixed glandular tumor, grown together with the skin, was found on the left side of the neck; small, movable glands were present on the right side. Between June 14 and July 16 the patient received a total of 7,750 r. On July 26 the tumor in the epipharynx had disappeared; the glands on the right side had nearly disappeared, and the glands on the left side were much smaller. On September 10 no tumor was visible in the epipharynx. In the neck one could touch thick scars, perhaps remains of glands. But the patient complained of a disturbance of the sense of taste and some dryness. On October 15 the disturbance of the taste had been improved; the left half of the tongue was atrophic, and in the epipharynx and on the neck the same conditions were present. On November 29 the findings in the epipharynx and on the neck had not been changed, but the patient had lost weight and had radiating pains. The roentgen findings in the paranasal sinuses and the base of the skull were normal. On Feb. 4, 1936, a local recurrence of the tumor in the epipharynx was found. On February 9 the patient received 296 mg. of radium and on May 27, 700 mg. On June 5 the respiration through the nose was improved and the tumor in the epipharynx diminished. Because of intense pains in the occiput the patient was admitted to the otolaryngologic department of the Policlinic in Vienna on July 6.

By that time the skin on the left half of the neck was atrophic, full of scars and drawn by ectatic blood vessels, the effect of treatment with roentgen rays. In that area some hard, fixed glands were present, reaching the supraclavicular fossa. There were a deviation of the nasal septum and some pus on the floor of the right half of the nose. The tongue was cracked, and the mucous membrane of the pharynx was dry and red. A tumor with a smooth surface filled the largest part of the epipharynx. The epiglottic folds on both sides were edematous, on the right side more than on the left; the motility of the vocal cords was normal, and the larynx was normal. The drum on each side was dull. An accentuated whispered voice was heard on both sides at 3 meters. On July 25 the roentgen findings at the base of the skull were normal; on August 16 the patient died.

Gross Observations at Autopsy.—The skin on both sides of the neck was strongly pigmented and atrophic. The subcutaneous tissue was callous in that area. The dura was only slightly stretched; the sinuses of the dura were normal. The vessels on the base of the brain showed arteriosclerosis of medium degree. The cut surface of the brain revealed much fluid; the ventricles were dilated and filled with clear fluid. In the left striated body of the brain some old softenings as big as a grain of pepper were present.

On the posterior wall of the epipharynx was a tumor as big as a walnut, with a smooth surface, bulged forward into the choana. Some necrotic shreds of the tumor, as big as a pea, could be observed at the superior margins of the choanae. The tumor had grown into the base of the skull (fig. 1), the sphenoid sinus, the left maxillary sinus and the cavity of the nose. In the posterior part of the left sphenoid sinus were mucous contents, with crumbs of necrotic tissue.

The tumor had grown into the clivus (fig. 1) and destroyed its right half as far as the internal cortical layer. The left half of the clivus was only less completely destroyed. Finally the tumor had reached both condyles of the occipital bone, destroying the right more than the left. The tumor could everywhere be sharply separated from the spongiosa of the marrow. The tips of the petrous bones were normal.

On the nasal surface of the soft palate was a sharply bordered, smeary ulcus as large as a nickel, with slightly elevated margins. In the mucous membrane of the left piriform sinus was a gray-white tumor as big as a lentil. The aryepiglottic folds were pale but edematous, more so on the right side than on the left. The vena jugularis on each side was accompanied by swollen glands, which made the lumen of the vein narrower.

Diagnosis.—The following conditions were present: a solid carcinoma of the epipharynx, which had progressed into the sphenoid bone, the left maxillary sinus

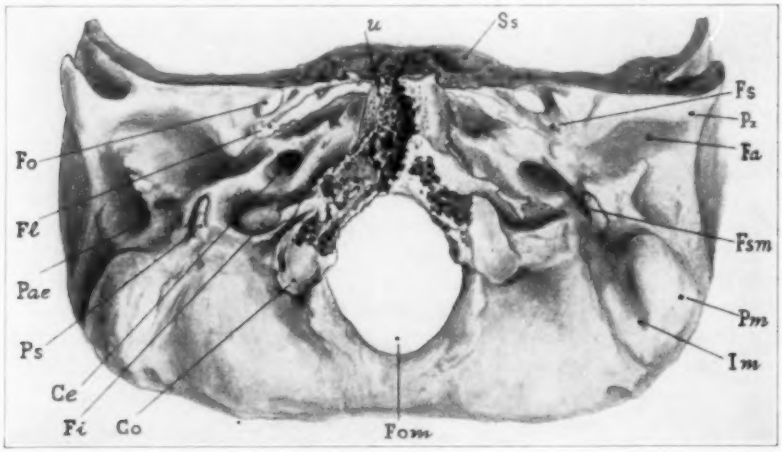

Fig. 1.—Base of the skull; *Fom,* foramen occipitale magnum; *Co,* condylus occipitalis: *Fi,* foramen jugulare; *Cc,* canalis caroticus; *Ps,* processus styloideus; *Pae,* porus acusticus externus; *Fl,* foramen lacerum; *Fo,* foramen ovale; *U,* ulceration on the base of the skull; *Ss,* sinus sphenoidalis; *Fs,* foramen spinosum; *Pz,* processus zygomaticus; *Fa,* fovea articularis; *Fsm,* foramen stylomastoideum; *Pm,* processus mastoideus; *Im,* incisura mastoidea.

and the nasal fossa; secondary carcinoma of the mucosa of the piriform sinus, the left lung and the left pleura; edema of both aryepiglottic folds; lobar pneumonia of the lower lobe of the right lung, partly receding; brown atrophy of the myocardium, and acute tumor of the spleen.

Microscopic Observations.—Soft Palate: On the nasal surface of the soft palate was a sharply bordered ulcus covered with pieces of necrotic tissue. The ground of the ulcus appeared to have been produced by a solid, partly necrotic carcinoma, which replaced the whole layer of the mixed glands and also broke through the infraglandular layer of elastic fibers. But the pegs of the carcinoma nowhere reached the muscle layer, whereas carcinoma cells could be seen in the lymphatic vessels in the neighborhood of the ulcus (fig. 2). It was remarkable that both surfaces of the soft palate, the nasal as well as the oral, appeared to

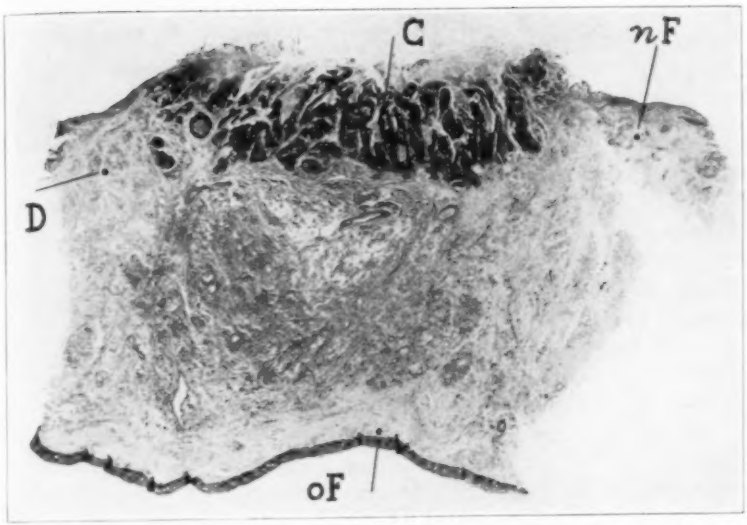

Fig. 2.—Carcinoma by contact (*C*) on the nasal surface of the soft palate (*nF*); *oF*, oral surface of the soft palate; *D*, layer of mixed glands; staining with hematoxylin-eosin.

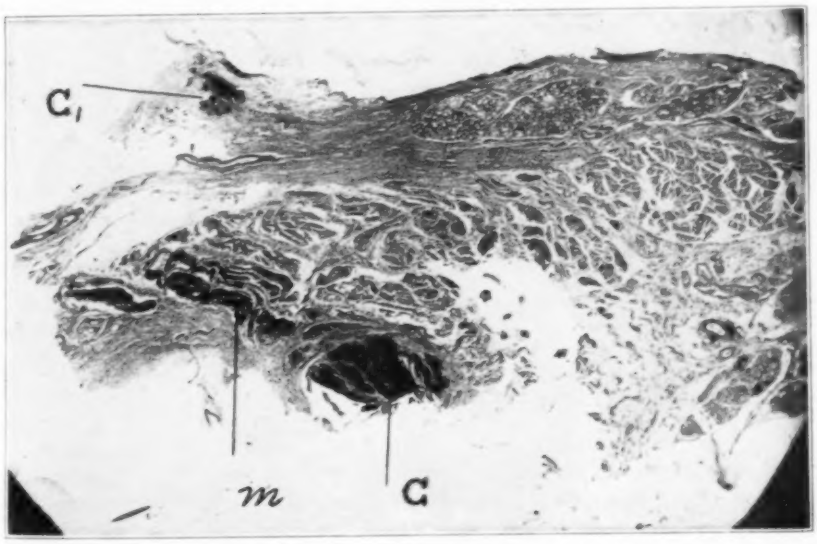

Fig. 3.—Mucous membrane of the lateral wall of the hypopharynx on the left side (staining with hematoxylin-eosin): *M*, muscle layer; *C*, metastasis of the carcinoma within the muscle layer; C_1, metastasis of the carcinoma within the subepithelial connective tissue.

be covered by squamous epithelium, which was thinner and without papillas on the nasal surface. A respiratory cylindric epithelium with its characteristic basilar membrane could not be seen in the surroundings of the ulcus.

Mucous Membrane of the Lateral Wall of the Hypopharynx on the Right Side: The epithelial layer was surprisingly flat in many places. Papillae were absent. Nowhere did the epithelium grow into the deeper layers. The layer of the lymphatic tissue was a little increased. Few perivascular infiltrations were to be noted.

Mucous Membrane of the Lateral Wall of the Hypopharynx on the Left Side: A solid carcinoma was embedded, infiltrating the striated muscle fibers in some places (fig. 3). In a few perivascular lymph spaces carcinoma cells could be seen. Many blood vessels were closed by thrombi.

COMMENT

The case described was of little interest from the clinical standpoint, since the patient was first seen in an almost hopeless condition. More interesting are the postmortem observations, of which I wish to stress: (*a*) the destruction on the base of skull, (*b*) the edema of the aryepiglottic folds, (*c*) the carcinoma in the lateral wall of the hypopharynx on the left side and in the left piriform sinus and (*d*) the carcinoma on the nasal surface of the soft palate.

(*a*) *Destruction on the Base of the Skull.*—The tumor had grown along the clivus backward as far as the foramen occipitale magnum, where it had partly destroyed the articulation between the atlas and the base of the skull. I wish to emphasize that the lamina interna of the clivus nowhere was perforated, although the tumor had invaded the greatest part of the clivus. This observation proves that the thin lamina interna was much more resistant against the invasion of the tumor than was the spongy bone of the marrow.

But this observation is interesting also from another point of view. A roentgenogram was made of the macerated specimen. The picture clearly showed the diploetic tips of the petrous bones, which were thoroughly normal. The bone of the clivus produced a fine shadow, which signifies a thin but normal bone. Hence, in comparison with the specimen itself, the roentgenogram gave a far from exact impression of the extension of the destruction within the clivus. Under these circumstances one surely cannot expect an exact picture of the base of the skull when the roentgenogram is made during life. A normal roentgen picture of the base of the skull in a case of carcinoma of the epipharynx, therefore, does not prove much.

(*b*) *Edema of the Aryepiglottic Folds.*—The edema might have had various explanations, including pressure from the swollen glands on the jugular veins, the thrombi within the mucous membrane of the pharynx, the cachexia, the bad action of the heart and finally the treatment with roentgen rays. Which of these factors was most important in this case cannot be ascertained.

(*c*) *Carcinoma in the Lateral Wall of the Hypopharynx on the Left Side and in the Left Piriform Sinus.*—It is not to be supposed that this case was one of multiple independent tumors, since the tumors in the hypopharynx showed the same structure as that in the epipharynx.

It is much more probable that some particles of the tumor in the epipharynx were deposited along the lymph vessels leading into the hypopharynx.

It is known that the lymph vessels of the pharynx produce a dense network. Although every part of the pharynx has its own chief means of drainage (Most, Bartels), the lymph vessels of the single parts of the pharynx communicate so extensively with each other that particles of a tumor can easily be delayed in passage from the epipharynx into the hypopharynx. Accordingly, in some lymph vessels of the mucous membrane of the pharynx carcinoma cells were noted. Since the carcinoma was situated in the deep layers of the mucous membrane, development of this tumor by inoculation from the saliva can surely be excluded.

This observation proves that sometimes with carcinoma of the epipharynx small metastases occur far away from the primary tumor within the mucous membrane of the pharynx, although this mucous membrane looks macroscopically normal. This must be kept in mind when the surgical treatment of malignant tumors of the pharynx is discussed.

Metastases in the lungs and in the pleurae are produced by the blood stream. They are not rare with carcinoma of the epipharynx (Graeff).

(*d*) *The Carcinoma on the Nasal Surface of the Soft Palate.*— It cannot be doubted that this was a typical carcinoma by contact. Many facts agree with that supposition. At first may be mentioned the extraordinary localization of the tumor. Carcinoma of the soft palate is not rare, but always it originates from the oral surface of the soft palate. On the contrary, the carcinoma in this case was situated on the nasal surface of the soft palate, which can easily be explained by the frequent contact between this surface and the tumor. Moreover, the carcinoma of the soft palate was localized within the superficial layers of the nasal surface and did not even reach the muscle layer. To summarize, the carcinoma of the soft palate was a carcinoma by contact, produced by the carcinoma in the epipharynx.

Finally, it is interesting that the tumor of the soft palate, which had the same structure as the tumor of the epipharynx, had already broken into the lymph vessels of the soft palate. Therefore, metastases could be produced by the tumor in the epipharynx as well as by the tumor within the soft palate.

The carcinoma of the soft palate is interesting also from the clinical standpoint. As is known, Trotter has described a triad of symptoms characteristic of malignant tumors of the epipharynx. One of these symptoms is restriction of motility of the soft palate. Trotter explained this symptom by a carcinomatous infiltration of the muscles of the soft palate. My case proves that this symptom can be produced also by a carcinoma by contact on the nasal surface of the soft palate.

IMPACTED FOREIGN BODY IN THE ESOPHAGUS

An Unusual Case

MARVIN L. LATIMER, M.D., CLIFTON SPRINGS, N. Y.

Foreign bodies in the esophagus offer many problems in diagnosis and in methods of extraction, nonopaque ones presenting especial difficulties. An impacted nonopaque foreign body in a mongolian idiot, inaccessible by peroral endoscopy, with a fatal result, makes this case worthy of report.

REPORT OF CASE

History.—A 12 year old mentally deficient boy was brought to the hospital with the following history: He was noted to be acting strangely a short while after supper. With his head bent forward, he was restless, drooling from the mouth and nose and having respiratory difficulty. Roentgenographic studies revealed the larynx to be displaced anteriorly, with no opaque foreign body in the esophagus. A foreign body was suspected, but no information was available as to its nature. The patient's diet consisted only of soft or macerated foods. Because of the mental deficiency, it was impossible to elicit subjective symptoms.

Physical examination revealed an idiot in acute distress, restless, drooling from the mouth and nose and obviously aspirating saliva with each inspiration. There was an occasional croupy cough. The patient held his head forward and resisted any type of examination. Positive findings included a palpable mass in the left side of the neck, posterior to and just below the cricoid cartilage. Moist rales were present over the trachea and larger bronchi.

In view of the history and physical findings, an esophagoscopic examination was indicated.

Operation.—Without anesthesia, the hypopharynx was exposed with a laryngoscope, and the mouth of the esophagus was found to be obliterated by a firm mass, which was apparently below the lip and firmly impacted between the cricoid cartilage and the vertebrae. There was marked edema of the mucosa. It was possible to move the mass in an anteroposterior direction with the tip of the laryngoscope and also laterally by bimanual manipulation. The swollen, redundant mucosa prevented a view of the foreign body. It felt "stony hard." The possibility of a congenital abnormality was considered. A bronchoscopic examination was done without difficulty, and a large amount of aspirated saliva was removed by suction. The mucosa of the tracheobronchial tree was inflamed and swollen. Further attempts at removal by peroral endoscopy, with the patient under ether anesthesia were unsuccessful.

The external approach was considered, but because the patient had demonstrable rales over both lungs and was cyanotic it was thought inadvisable. Bronchopneumonia rapidly developed, and the patient died within twenty-four hours, despite constant throat suction and the use of oxygen.

From the Clifton Springs Sanitarium and Clinic.

Autopsy.—Other than the cast of features which tended to confirm the diagnosis of mongolian idiocy, there were no abnormalities on the surface of the body. Examination of the thoracic and abdominal organs revealed no gross abnormalities except that the lungs were heavy and dark red. On section, a considerable amount of dark red fluid escaped, and the cut surfaces were everywhere studded with dark red consolidated areas, the largest of which measured 10 mm.

After the viscera had been removed, embalming fluid was injected into the carotid arteries, and the organs of the neck were removed en masse. The trachea was patent, and the mucous membrane was deeply injected and partially covered

Foreign body (radish) in the esophagus. Posterior view.

with mucus. The mass could be felt at the upper end of the esophagus, immediately behind the lower end of the thyroid cartilage. Viewing the specimen from above, one observed marked edema of the epiglottis and surrounding mucous membranes, so marked, in fact, that the upper end of the esophagus appeared completely obstructed. Attempts to pass probes into the esophagus were entirely unsuccessful, a firm obstruction being met at every point. The esophagus was opened by means of a knife, with an incision from without inward. So great was the tension that immediately on section the walls retracted, in spite of the partial fixation by the embalming fluid. The foreign body thus brought into view proved to be a radish, slightly flattened, spheroid and measuring 2.5 by 2.5 by 2 cm. When

this was lifted from its bed, the mucous membrane was seen to be a mottled red and greenish yellow. The posterior wall of the esophagus measured not more than 1 mm. in thickness.

Microscopic Examination.—Sections from a block taken at the level of the foreign body, which included both esophagus and trachea, showed almost complete destruction of the esophageal epithelium. There was some necrosis of the submucosa and at places bacterial invasion. Leukocytes were conspicuously absent at this level. Sections taken from the tissues at the margin of the foreign body showed marked edema of the tissues and some infiltration with polymorphonuclear cells. On the tracheal side of the section the epithelium was intact. There was injection of the blood vessels of the mucosa and considerable infiltration with lymphocytes and plasma cells but only a few polymorphonuclears.

Sections from the lungs showed an early stage of bronchopneumonia, with a large number of red blood cells in the alveoli, marked edema and here and there rather large numbers of polymorphonuclears; in addition, there were many clumps of bacteria scattered throughout the sections.

Sections from the other organs revealed nothing worthy of note.

Anatomic Diagnosis.—A foreign body (radish) was lodged in the esophagus, causing complete obstruction, necrosis and marked edema of the mucosa; there was acute bronchopneumonia (aspiration).

COMMENT

This case has been presented to report an unusual type of foreign body (radish) firmly impacted in the esophagus and impossible to remove by the peroral route. To add to the difficulties of diagnosis and treatment, the patient was a mongolian idiot, which eliminated subjective information and essential facts of the history. Death was due to a drowned lung because of the obstruction of the esophageal opening with an overflow into the trachea.

NASOPHARYNGEAL ULCERATION RESULTING FROM PRESSURE NECROSIS

ARTHUR G. FORT, M.D., AND LESTER A. BROWN, M.D.
ATLANTA, GA.

In this article we report a case of ulceration of the posterior pharynx caused by pressure necrosis due to a sharp-pointed anterior tubercle of the atlas.

REPORT OF A CASE

E. B., a 17 year old white girl, was referred to us from another city on Nov. 27, 1937, with the complaint of spitting up blood-streaked sputum. The history was that she awoke one morning in October 1937 with a sore throat, which gave her little concern until forty-eight hours later, when she was noticeably worse and hoarse. The school nurse found that there was some blood in the back of the throat and put her under the care of a local physician. He noted that she had a temperature in the afternoon of from 99.5 to 101 F. (mouth) and had her confined to bed, but while in bed she had no fever. She was suspected to have tuberculosis, and therapy was instituted on that basis but with entirely negative results. Her history showed that she had had chickenpox, whooping cough and measles as a child, bronchitis in 1936 and malaria in 1937, without complications. On July 26, 1925, when the patient was 5 years old, she was struck by an automobile in Atlanta, caught by the left foot and dragged, face down, for approximately 50 yards (46 meters). She sustained a laceration of the lower jaw and an injury to the leg, which necessitated treatment for about one year, since which there had been no deformity or loss of function. So far as she knew, there was no injury to her neck.

On examination in our office on Nov. 27, 1937, she was seen to be a rather tall (5 foot 4 inches [163 cm.]), slender girl, weighing 105 pounds (47.6 Kg.). Her temperature was 98.6 F. (mouth). Her eyes, ears and nose showed no pathologic signs. Her teeth were good. Her tonsils had been removed. There were some streaks of blood in the posterior pharynx, apparently coming from above the level of the oropharynx. By elevating the soft palate, there could be seen on the surface of the posterior nasopharynx, just over the region of the first cervical vertebra and slightly to the left of the midline, a superficial ulceration about 1 cm. in diameter. It appeared that the mucous membrane was denuded here and that the bleeding was coming from this area, which surmounted a prominence of the posterior pharyngeal wall, more to the left than to the right side. This prominence on palpation was felt to be bone and moved as the atlas moved. With the finger on this point, when the patient swallowed one could feel the pharynx slide over this stationary bony prominence. A specimen for biopsy was taken, as was blood for a Wassermann test, and the patient was sent to the Winship Clinic of the Emory University Hospital for roentgen treatment of the posterior pharynx. Dr. J. E. Scarborough, of this department, noted the findings described and had a lateral roentgenogram made of the neck. The

report given by Dr. J. J. Clark was: "There is an anterior curve to the cervical spine, which is probably the result of bowing in the lumbar region. The head is tilted forward on the first cervical vertebra, projecting the tubercle of the atlas prominently into the posterior pharynx. There is no evidence of a pathologic condition of bone, nor is there deformity of the nasopharynx." The biopsy was reported by Dr. E. L. Bishop as follows: "Chronic infection of unknown origin is present. There is no evidence of neoplasm or tuberculosis in this specimen." The Wassermann reaction was reported to be negative. Roentgen therapy was given, and the bleeding stopped for about three weeks, during which time the patient returned to school, but she came back to us in March 1938, with the same complaint, and examination revealed the same findings as before.

On March 9 she was admitted to the otolaryngologic service of Grady Hospital for a complete treatment. Her temperature was 99 F. (mouth) on admission and occasionally went to 99 F. during her stay in the hospital but was for the most part normal. The blood count was: hemoglobin 80 per cent, erythrocytes

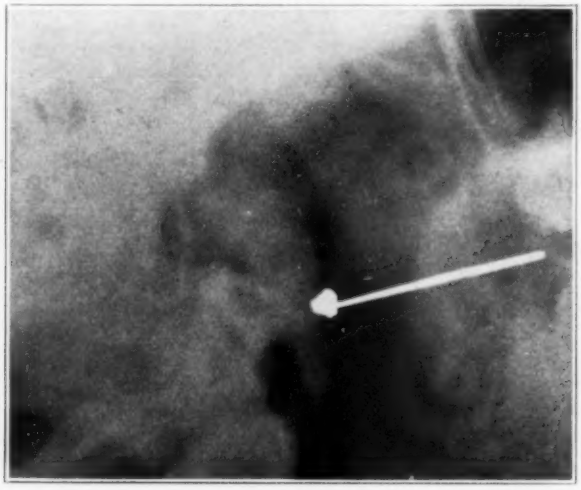

Fig. 1.—Preoperative roentgenogram, showing the sharp-pointed, large anterior tubercle of the atlas.

3,440,000, leukocytes 7,800, polymorphonuclears 80 per cent, lymphocytes 20 per cent, platelets 206,500, coagulation time two and one-half minutes (capillary tube), bleeding time three and one-half minutes. Chemical analysis of the blood showed sugar 111.1 mg. per hundred cubic centimeters, nonprotein nitrogen 31.5 mg. and creatinine 1.4 mg. Urinalysis showed a trace of albumin and 3 to 5 leukocytes per high power field. Culture from the throat showed streptococci. The Wassermann reaction was again negative. Examinations for tuberculosis, including cutaneous tuberculin tests, gave negative results. Smear for malaria was negative. Physical examination, except for that of the pharynx, gave negative results. Lateral roentgenograms of the neck showed a prominent anterior tubercule of the atlas, with this structure coming to a point anteriorly; that is, it appeared as a wedge with the base against the vertebra (fig. 1).

On April 1, with the patient under ether anesthesia, the mouth and pharynx were mopped with an aqueous solution of metaphen, after which tincture of

metaphen was applied over the operative field. A solution of procaine hydrochloride and epinephrine was injected in the region of the ulcer for hemostasis and an incision was made vertically through the denuded area over the bony prominence. The soft tissue was divided, and the anterior ligament and the longus colli muscle were separated from the tubercle, which was then found to be sharp and projecting slightly to the left from its origin. With bone rongeurs and curets, this point was shaved off slowly until the tubercle had a rounded, smooth surface (fig. 2). One large vein was tied with black silk, and the incision was closed with black silk, the lower fourth being left open for drainage. The postoperative course included a marked swelling of the tissues of the posterior pharynx, with some dysphagia and some pain on moving the head. The temperature was 99.5 F. on the first postoperative day, 99 F. on the seventh and 100 F. on the ninth, but otherwise completely normal. After the fourth day, warm saline irrigations of the nose and throat were used for cleansing the nasopharynx. The operative field healed slowly but progressively, and on May 30

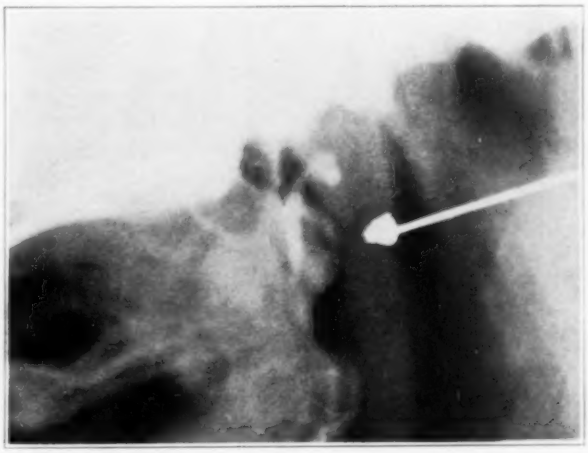

Fig. 2.—Large, sharp-pointed tubercle of the atlas (fig. 1) shaved off to a smooth, rounded surface.

the patient was pronounced completely healed, the only indication of the previous pathologic condition being a small scar in the nasopharynx, covered with normal mucous membrane.

In looking through a number of lateral roentgenograms of the neck, taken serially from the files of Grady Hospital, we were surprised to find that many of them showed prominent anterior tubercles, several being fully as prominent as that of the patient; however, none of the patients had had a similar complaint. Then, through the cooperation of Dr. Homer Blincoe, professor of gross anatomy, Emory University School of Medicine, we were able to examine many dried specimens of the atlas, and here again we found a wide variation in different specimens, from the atlas with almost no anterior tubercle to that with a tubercle which was extremely large, even having a beak-like projection on its lower extremity hanging over the axis. It was not uncommon, and in fact it seemed rather the usual thing, for the anterior tubercle to be prominent. One difference appeared uni-

formly, however; in the many specimens and roentgenograms studied no tubercle showed the marked pointing anteriorly of that in our case. Macalister,[1] in the latter part of the nineteenth century, in a treatise on the atlas said of the anterior arch:

> Its thickness . . . varies in the centre, owing to the varying size of the anterior tubercle. . . . [In one specimen] this is large, and projects 8 mm. in front of the arch. . . . [In another] it projects downwards in front of the axis. This latter condition is due to the extension of ossification into the attachment of the anterior ligament. . . .
>
> On the other hand, the tubercle may be, but very rarely, obsolete, and the arch diminished to 5 mm. in thickness. . . . The form of this tubercle also somewhat varies, and rarely shows some tripartition, the median part being for ligamentous, the lateral for muscular attachment. . . .
>
> [There may be] upward extension of the tubercle, ossification extending along the middle slip of the atlanto-occipital ligament.

He stated that the tubercle may even articulate with the occipital bone.

CONCLUSIONS

We have presented a case of ulceration of the posterior nasopharynx caused by pressure necrosis due to a sharp-pointed, large anterior tubercle of the atlas.

The deformity of the anterior tubercle possibly was caused by injury to the anterior arch, producing impingement against the dens, in an automobile accident in 1925.

We have not been able to find a similar case reported in the literature and therefore regard this type of ulceration as rare.

Drs. James King and Earl McKey, interns of the otologic, rhinologic and laryngologic service of Grady Hospital assisted. Miss Mildred Jordan, librarian of Emory University School of Medicine, searched the literature.

478 Peachtree Street Northeast.

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1. Macalister, A.: Notes on the Development and Variations of the Atlas, J. Anat. & Physiol. **27**:519-542, 1892-1893.

Clinical Notes

PHYSIOLOGY OF HEARING

Historical Note

HARRY FRIEDENWALD, M.D., BALTIMORE

In his admirable work on the history of otology, Politzer[1] was able in a few pages to give a detailed account of all that was known about the anatomy and the physiology of the organ of hearing from the time of Hippocrates until the Renaissance. It was scant. Nothing was found in Hippocratic writings concerning the anatomy, and the physiology was summed up in the statement that "sound passing into the ear and penetrating the dry and delicate membrane resounds in the empty spaces of the hard bone."[2] In the millenium and a half that followed Hippocrates, the distinction between the auditory and the facial nerves was discovered (Galen); the organ of hearing was described as "composed of the petrous bone, the outspread auditory nerve, . . . and pent-up air, and of the diverticula and tortuous windings inside the opening, and of the external cartilage which can be seen around the opening like a shell" (Bernard de Gordon in 1305).[3]

The ossicles were not mentioned definitely until the early part of the sixteenth century, when Berengario da Carpi (in 1521) described the membrana tympani, which he still considered an expansion of the auditory nerve; he stated that "the empty space closed by the membrane contains implanted air, which takes up the various sounds and transmits them to the auditory nerve dilated into this membrane . . . two little ossicles are placed on this membrane, so arranged that they are moved by the movement of the adjacent air and that by their movement they react on each other."[4] Massa (in 1536) described the one ossicle as like a drumstick and therefore called malleolus, and stated that it is movable, attached to and moving with the drumhead. But he also stated that these ossicles were discovered in the latter part of the fifteenth century.[5] The long period of stagnation has now come to an end. Vesalius, Ingrassias, Fallopius and Eustachius were the heralders of the renaissance of otology. The historical accounts have been collected by Politzer and more fully still by Morgagni.[6]

In his "Fabrica" (in 1543) Vesalius described the method of dissecting the structure of the ear and the importance of studying the anatomy of the ear in animals; he described the malleus and the incus and pictured them, the two fenestrae and the tensor tympani muscle (though he did not recognize its muscular nature).

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1. Politzer, A.: Geschichte der Ohrenheilkunde, Stuttgart, Ferdinand Enke, 1907, vol. 1.
2. Hippocrates, cited by Politzer,[1] p. 15.
3. Bernard de Gordon, cited by Politzer,[1] p. 54.
4. Berengario da Carpi, cited by Politzer,[1] p. 75.
5. Massa, cited by Politzer,[1] p. 76.
6. Morgagni, J. B.: Opera omnia, Patavii, ex typographia Remondiniana, 1765, vol. 1, pts. 2 and 3, Epistolae anatomicae 12 and 13, pp. 117 and 144.

In his second edition (1555) he likewise made no mention of the stapes, but in his "Examen" of the observations of Fallopius in the famous letter to the latter published in 1564 he admitted that it was "overlooked" by him (Verum interim in tuis observationibus quoddam istius organi adducis ossiculum a me neglectum)[7] and gave credit to Ingrassi as for the discovery. Concerning the physiology of hearing Vesalius wrote with scientific modesty: "There is nothing about hearing and the perception of sound that I can add."[8]

The ear had now become an object of interest and study. There is a goodly list of those who made great advances in the knowledge of its anatomy. Foremost among them were Fallopius (earliest publication in 1561) and Eustachius (earliest publication in 1563).

These advances in anatomy could not fail to lead to speculation as to the function of hearing. Morgagni gave a detailed account.[6] Concerning Fallopius he wrote: "This much is certain, that before all the others whom we have mentioned he observed that when the myringa is agitated or percussed then the hammer is moved, with the incus and the stapes, or that after the cavity has been opened when one of these bones has been agitated by a needle, then the two others at the same time join in the movement."[9] Morgagni added: "If he had known, moreover, that the basis of the stapes is connected with the fenestra ovalis through a membrane so that it can be moved only slightly he would probably have come to the conclusion which as I see is now adopted by scholars . . . ."

Fallopius' "Observations" appeared in 1561. It is this fact that has led to the publication of this note. For in one of the most interesting cases which Amatus Lusitanus published in his seven "Centuriae," there was an account of a legal trial in Ragusa, in which a woman was imprisoned and tried on the charge of causing a young man to become deaf by witchcraft. The judges called on Amatus as an expert, and his case history contains the report he made, by which he convinced the court that the deafness was due to syphilis. In this report occurred the following remarkable statement: "The organ of hearing is of such delicacy that it is injured by the slightest cause and led to develop this very disease, for the bony stapes, as I have said elsewhere, as well as the incus bone, on which the malleolus strikes, are held together by ligaments; when moved by sounds, the malleolus strikes the anvil. Then the (animal) spirits existing in the nerves are excited by this percussion and from this hearing results."

The exact date of the trial is not known. Amatus was in Ragusa from 1556 until some time in 1559, when he went to Salonica. The volume of "Centuriae," in which the case appeared was finished in 1559 and was published in 1560.

This description of the function of the ossicles is remarkably like that of Fallopius, which appeared, as has been said, in 1561.

It is not my intention to lay claim to priority for Amatus, but rather to show that this physician, who was a careful and brilliant student, interested in the great progress of the Renaissance, had not allowed even these very recent discoveries to pass by without careful note. It is not improbable that these studies,

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7. Vesalius, A.: Opera omnia and anatomica et chirurgica, edited by H. Boerhaave and B. S. Albinus, Lugd. Bat., J. du Vivie et J. et H. Verbeek, 1725, p. 770.

8. Vesalius, A.: De humani corporis fabrica libri septem, Basileae, ex. off. J. Oporini, 1543, p. 650; cited by Politzer,[1] p. 85.

9. Morgagni;[6] cited by Politzer,[1] p. 94.

made in Italy, became known to colleagues and other scholars before they appeared in print. Amatus spoke with high admiration of Fallopius [10] and Eustachius [11] and appears to have had personal acquaintance with both.

This account of Amatus was not the only surprise that this subject furnished me. While I was writing out these notes, my attention was called to the following paragraph in Aldous Huxley's "Point Counter Point": "The shaking air rattled Lord Edward's *Membrana tympani*; the interlocked *malleus, incus* and stirrup bones were set in motion so as to agitate the membrane of the oval window and raise an infinitesimal storm in the fluid of the labyrinth. The hairy end imps of the auditory nerve shuddered like weeds in a rough sea; a vast number of obscure miracles were performed in the brain and Lord Edward ecstatically whispered, 'Bach!'" [12]

When such facts make their appearance in popular novels, it is proof that what was once a great discovery has become popular science.

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10. Amatus, L.: In Dioscoridis Anazarbei de medica materia libros quinque Venice, Scotus, 1553, book 2, enarratio 172; book 3, enarratio 78. The name appears as Gabriel Mutinensis, but the index shows this to be "Gabriel Falopius Mutinensis."

11. Amatus, L.: Curationum medicinalium, Centuria I, Curatio 1. This book appeared in many editions; reference is made here to the numbers of the centuria and the curatio concerned, which are the same in all editions. The section mentioned may be found by consulting any edition that contains the first 100 cases.

12. Huxley, A.: Point Counter Point, New York, Doubleday, Doran & Company, Inc., 1928, p. 32.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

ANATOMY AND PHYSIOLOGY OF THE EAR

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In reviewing the literature from October 1937 through September 1938 pertaining to the anatomy and physiology of the ear, we present summaries and abstracts of articles that permit the reader in a brief time to obtain a panorama of the field concerned. The review is not intended to be self sufficient as are the original articles. Translations of foreign literature were made when the articles were of special value.

DEVELOPMENTAL ANATOMY

In another contribution to the study of the development of the otic capsule Bast [1] describes the fossula post fenestram, which is similar to the fissula ante fenestram described previously by him.

He has observed that

> The fossula post fenestram is an evagination of the periotic tissue extending into the bony capsule just posterior to the oval window, at a point about one third of the way between the oval window and the nonampullated end of the lateral semicircular canal. It may be compared with the fissula ante fenestram in character and content. It differs from the fissula ante fenestram in that its shape is more sacklike and that as a rule it opens only from the vestibule and does not extend to the middle ear, although in a few cases it does communicate with both the vestibule and the middle ear. The fossula post fenestram is not a constant structure. . . .
>
> . . . the precartilage in this area dedifferentiates into a loose mesenchymal type of tissue, which later becomes slightly fibrous. This fossular tissue in most cases communicates with the vestibular tissue as it does in the fissula ante fenestram, but it differs from the latter in that it opens into the middle ear in only a small percentage of cases. When it does open into the middle ear, it receives blood vessels from the middle ear, as is the case in the fissula ante fenestram.

1. Bast, T. H.: Development of the Otic Capsule: Fossula Post Fenestram. Arch. Otolaryng. **27**:402 (April) 1938.

He describes the various anatomic types of fossula. These are purely developmental and are not functional.

Detailed observations have been made by Martin and Anson[2] on sections of the otic capsule and membranous labyrinth of a 29 mm. (crown-rump) human embryo (estimated to be 1.87 months of age). This article is a first study "in a projected group dealing with the development of the cartilaginous otic capsule and contained membranous labyrinth in human embryos." Reconstructions were made of the capsule alone, without surrounding structures, so that its more familiar landmarks might be shown and the contour and relation of its parts studied. The authors state:

> There is no indication at the 29 mm. stage of separation of the head of the malleus from Meckel's cartilage. . . . The manubrium of the malleus is an elongate structure lying inferior to Meckel's cartilage and arising from the neck of the ossicle at an angle of approximately 60 degrees with the cartilage. . . .
>
> . . . The stapes is essentially annular; it is lodged in loose cellular tissue, and through it pass small blood vessels, although the stapedial artery has not yet made its appearance. . . .
>
> The [oval] window at this stage is not oval; it is a roughly quadrilateral opening, no two sides of which are of equal length. . . .
>
> The membranous labyrinth, as seen in the reconstructions, presents many features characteristic of the adult condition. The cochlear duct, made up of two and a quarter turns, is considerably flatter (that is, less conical in cross section) than it is in the adult ear; yet a transverse section of the duct already shows its general triangular shape, with the apex of the triangle pointing toward the modiolus. The semicircular ducts, with their ampullae, and the common crus approximate closely the adult form. . . .
>
> The semicircular canals have relationships to one another similar to those of the corresponding ducts. Ampullary dilatations are already discernible. . . .
>
> Mesenchymal vacuolation in the areas later to be occupied by the large periotic spaces has begun in the 29 mm. embryo. Multiple small disconnected cavities have appeared, some of which are vascular spaces lined by endothelium while others are true forerunners of the periotic spaces, certainly identifiable only where the loose lining cells have united into a thin but regular membrane. . . .
>
> There is some suggestion of papillary folds on the canalicular epithelium, but this is more marked, though still primitive, in the epithelial walls of the utricle and the saccule. The valve at the utriculo-endolymphatic communication is well formed. The entire valvular mesenchymal tissue is conspicuously vascular. . . .
>
> The fissula ante fenestram is not yet present . . .; no budding of the vestibular space toward the tympanic cavity is to be seen anywhere in the region anterior to the vestibular window, nor is there indentation suggestive of a tympanic orifice. There was no hint of differentiation of tissue which would suggest the early formation of the fissula post fenestram. . . .
>
> The stapedial muscle, still rather indefinite at its proximal end, arises just anterior to the mastoid cartilage and passes medially under the styloid process;

2. Martin, J., and Anson, B. J.: Otic Capsule and Membranous Labyrinth of the Twenty-Nine Mm. (Crown-Rump) Human Embryo, Arch. Otolaryng. **27:** 279 (March) 1938.

where it passes just medial to the facial nerve it is a well developed structure. Since there is no pyramid at this stage, the tendinous attachment to the posterior surface of the neck of the stapes is more definite than is that to the capsular wall. The tensor tympani muscle arises from the cartilaginous portion of the auditory tube; throughout its length this muscle is much larger than the stapedius. It passes backward along the medial tympanic wall and lies free in the mesenchyme of the tympanic cavity, since the semicanal is not yet formed. It attaches laterally to a low tubercle on the manubrium.

The elastic fibers of the tympanic membrane were investigated by Zanzucchi.[3] He has examined tympanic membranes removed within twenty-four hours after death from 27 subjects, including fetuses and persons from 1 to 75 years old.

In the fetal stages the membrane is comparatively thick and vascularization is considerable. The radial fibers are well marked, especially in the membrana tensa, while the circular fibers are less obvious.

In the newborn child, the membrane is less thick, and there is less vascularization, the radial and circular fibers occurring in the same proportion.

In subjects from 1 to 8 years of age, the vascular tissue becomes progressively less marked, and the fibers are more obvious and are attached to the periosteum of the malleus and the tympanic ring. Up to 8 years of age the fibers are found to consist chiefly of collagen, their elastic nature being only occasionally seen.

From 9 to 15 years, the fibers take the stain of elastic tissue and begin to show their adult appearance. The radial fibers reach from the handle of the malleus to the tympanic ring, and the circular ones are well marked in the region of the umbo. Elastic fibers are present in the membrana flaccida and show their elastic characteristics a little earlier than those in the membrana tensa.

From 15 to 25 there is not much change except that the fibers are a little more obvious, but from 26 to 50 the classic arrangement in two zones of radical and circular fibers is seen.

From 51 to 75, the substantia propria becomes thinner, and there is a deposition of sclerosing fibers around the umbo and at the periphery. At this stage the elastic fibers are much less obvious and take the specific stain well.

According to Wittmaack [4] the structural type of the mucosa of the middle ear is characterized by the histologic architecture of its components, the epithelium, the subepithelial tissue and the vascular system. He considers hereditary as well as external influences to determine

3. Zanzucchi, G.: On the Modification of the Elastic Fibers of the Tympanic Membrane at Various Ages, Arch. ital. di otol. **50**:203 (April) 1938.

4. Wittmaack, K.: The Development of the Structural Types of the Middle Ear Mucosa, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **29**:530, 1938.

the structural type in each person. Four such types are recognized: the mesoplastic, the hyperplastic, the hypoplastic and a combination of the two last. The terms hyperplastic and hypoplastic are here used with reference to the prevailing amount of fibrous tissue. If the mucosa is not subjected to unusual external influences, the mesoplastic type will prevail. The other type will be conditioned by the character, intensity and duration of unusual external influences. Wittmaack takes issue with Albrecht and Schwarz, who do not recognize external influences as determining factors. He doubts the validity of Schwarz' studies of the embryonic development of the mesenchymal elements. On the other hand, he considers changes in the chemical constitution and the hydrostatic pressure of the amniotic fluid important external influences. Schwarz, on the other hand, maintains that his work on the embryonic development of the mesenchymal elements has in the meantime been substantiated by extensive anthropometric studies and that on the whole Witmaack's views regarding extrinsic influences are no longer shared by the majority of workers in this field.

Anson and his co-workers [5] have made a reconstruction by the Born waxplate method of the petrous pyramid of a 4½ year old child. In this child the pneumatic pattern was seemingly established. They found the pneumatic cells of the petrous pyramid to

originate from two sources: (*a*) from the hypotympanic space—tubal cells; (*b*) from the extension of the mastoid cells (epitympanic cells).

The tubal cells are wide-spread, originate near the tympanic orifice of the Eustachian tube, and lie chiefly in the lower arc of the pyramid. They are in close proximity to the carotid canal with its important contents. This relationship of the tubal cells to the carotid canal was noted by Bezold and Siebenmann [6] [in 1897, when they made the first careful study of the tubal cells, based chiefly on a corrosive specimen. This] . . . reconstruction . . . brings out the difficulty of surgical approach to these cells, as compared with the case of opening the epitympanic cells by the mastoid route. Further, it makes clear how the disease might persist in a restricted area while the tympanic cavity and mastoid became relatively free from disease; the resulting improvement of hearing would then lead to a feeling of security both false and deceptive. In the child the preponderance of marrow cells over air cells in the pyramid may, through the phagocytic power of the marrow cells, act as a deterrent to the spread of petrositis.

In [the] . . . sections the pneumatic cells of mastoid origin are larger than the tubal cells. They are epitympanic and approach the lateral side of the internal auditory meatus. They lie in close proximity to the dura, in many cases separated from it only by a thin plate of bone. Disease in these cells, however, is relatively easy to approach from a mastoid opening; but the difficulty of opening the tubal cells is evident from their distribution and from their proximity to the carotid canal.

5. Wilson, J. G.; Gaardsmoe, J. P., and Anson, B. J.: Air Cells of the Petrous Portion of the Temporal Bone, J. Laryng. & Otol. **52**:746 (Nov.) 1937.

6. Siebenmann, F.: Anatomie des Mittelohres, in von Bardeleben, K.: Handbuch der Anatomie des Menschen, Jena, Gustav Fischer, 1897, vol. 5, pp. 237-238.

Adolph Rainer [7] states:

In completely normal pneumatization of the mastoid process the formation of cells in the petrous pyramid is not the rule. It is rather a kind of excess pneumatization and depends on a specific vital expression of the pneumatizing mucosa. The development or nondevelopment of cells in the pyramid is determined by the hereditary plastic property of the mucosa of the middle ear, which varies in each person, and by the structure of the bone, which is also subject to wide individual variations. (Formation of extensive tracts of cells can only occur when a sufficient number of marrow spaces is present in the bone of the petrous pyramid.)

The upper and the posterior group of cells, as well as the tractus subarcuatus, originate in the antral region, while the lower and the anterior group of cells are continuous with those in the floor of the tympanum, i. e., from the region of the tube. Formation of the cells is started when the layer of connective tissue of the mucosa increases, at times with considerable hyperemia, and proceeds in the direction of a neighboring marrow space, resorbing the intermediary bony wall. When this reabsorption is complete, the connective tissue penetrates into the marrow space and replaces the bone marrow. In the second phase, epithelium grows in, and the connective tissue regresses until a new cell, with a normal lining of mucosa, is formed. Further formation may take place after the manner of interstitial formation of cells. However, extensive pneumatization of the pyramid solely on the basis of this interstitial formation does not occur. In the walls of the already completed cells, destructive processes are often observed, which produce a smoothing of the walls as well as an increase in the size of the cells. Destructive phenomena in the bone, as for instance in the osseous wall of the hiatus subarcuatus, favor the process of pneumatization.

This process in the pyramid begins after the third year of life and may continue until old age. Therefore the most extensive pneumatization of the pyramid is found most often in old subjects.

Protracted suppuration in the pyramid may fill the cells with new-formed bone. The inflammatory processes in the bone marrow which accompany any protracted suppuration of the cells of the pyramid lead to its transformation into fibrous marrow. New formation of bone also takes place in the fibrous marrow spaces and may result in extensive sclerosis by which the further course of pneumatization is unfavorably influenced.

Rainer [8] also describes his microscopic study of the subarcuate fossa in 100 petrous pyramids, 20 of them fetal. He found that the subarcuate fossa is largest from the sixth to the eighth month of fetal life. It is filled with a plug of connective tissue. Ossification of this plug proceeds from the periphery, the finer structure of the bone undergoing numerous changes. At the fifth year, the fossa has been reduced to what the author calls the subarcuate hiatus. In the region of the latter, bone is laid down until the twentieth year. The author contrasts this protracted ossification with the absence from earliest youth of changes in the bone of the enchondral labyrinth capsule.

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7. Rainer, A.: Development and Construction of Pyramidal Cells, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **145**:3 (July 14) 1938.

8. Rainer, A.: The Ossification of the Subarcuate Fossa, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **29**:603, 1938.

Hardy[9] states: "The length of the organ of Corti is the distance from its base to its apical end as measured along the curves of the cochlear spiral. . . . The literature contains records for but nine human ears." Hensen (1865) was the first to make measurements. Hardy measured 68 human cochleas by Guild's method of indirect graphic reconstruction. She states:

> The age range is from 10 weeks to 85 years. . . .
>
> The average total length, measured along the line of pillar cell junctions, is 31.52 mm. The average lengths of the turns are: basal, 18.23 mm., or 57.9% of total length; middle, 9.28 mm., or 29.4%, and apical, 4.01 mm., or 12.8%.
>
> The variation is about 10 mm., from a minimum of 25.26 mm. to a maximum of 35.46 mm. . . .
>
> Bilateral symmetry of the organs of Corti in an individual is demonstrated. . . .
>
> The number of turns is from two and a half to two and three-quarters in . . . 87% of the cochleae. . . . [None had three turns and the shortest had two and one-sixth turns.]
>
> In the male the organ of Corti is on an average 3% longer than in the female.
>
> The data indicate a slight increase in total length of the organ of Corti with advancing age, but no change in the number of turns.

Wever[10] gives the following information:

> The width of the basilar membrane was measured in 25 human cochleas at a number of positions from base to apex. By the graphic reconstruction method, these positions were determined for each cochlea in terms of the distance from the basal end.
>
> The measurements for the different ears were in good agreement in the basal part of the cochlea, but showed wide variations in the apical part. Most of the basilar membranes, and particularly those of more than average length, attained a maximum width at a point about a half-turn before the apical end, and then decreased rapidly.
>
> The average value of the maximum points measured was 498 micra, which is about six and one-fourth times the minimum width of 80 micra as measured in a special series of sections.
>
> No significant relation was found between the form of the basilar membrane and age, sex, or race, or the condition of hearing as shown by audiometer tests made before death.
>
> It is pointed out that a theory of hearing that postulates a differentiating role for the width of the basilar membrane must allow for a wide range of normal variation in the apical region.

The appearance of the peripheral endings of the cochlear nerve on the hair cells of the organ of Corti of guinea pigs is described by

9. Hardy, M.: The Length of the Organ of Corti in Man, Am. J. Anat. **62**:291 (Jan.) 1938.

10. Wever, E. G.: The Width of the Basilar Membrane in Man, Ann. Otol., Rhin. & Laryng. **47**:37 (March) 1938.

Covell.[11] Supervital staining clearly demonstrates degenerative changes in the hair cells and nerves in animals injected with quinine.

Degeneration of the myelin sheath of the cochlear nerve fibers when produced by experimental methods is usually first apparent for the fibers in the osseous lamina spiralis [sic] and modiolus of the cochlea.[12] Similar changes appear later in the fibers of the auditory nerve. The effects of various agents such as drugs, bacterial toxins and missing dietary factors should be correlated with histopathological changes in the cochlear nerve and other lesions in the different parts of the peripheral auditory mechanism.

In an earlier report [12] by Covell and Noble appears the following passage:

An attempt to determine the significance of certain histopathological changes within the various structures of the cochlea and the cochlear nerve has resulted in the use of different experimental agents. A cytological study of the effects of drugs on the cochlea [13] has shown the external hair cells, the external sulcus cells and also those of the stria vascularis to be susceptible to the action of such drugs as quinine and sodium salicylate. The present study is a continuation of the drug effects and also includes the effects of a bacterial toxin and certain dietary factors on the cochlear nerve. Observations have been made chiefly on the changes in the myelin sheath of this nerve, using a modification of the Marchi method. . . .

Experimentally the changes are more pronounced in the nerve within the basal whorls of the cochlea and peripherally. Demyelinization gradually extends towards the middle and apical turns and finally involves the nerve fibers within the modiolus. The process thus extends from the periphery towards the more central pathways as has been shown by changes in the auditory nerve with its fibers from the cochlea and vestibule.

Animals which received quinine and salicylate for six to ten weeks showed extensive degenerative changes. A staphylococcus toxin administered to kittens in several injections and over a period of several weeks resulted in drastic changes within the myelin sheath. Various vitamin deficient animals including avitaminoses A, B_1, B_2, B_6, "filtrate factor" and D showed varying grades of changes within the myelin sheath which could be correlated with the severity of symptoms produced and the length of time the animals received deficient diets. Evidence tends to point towards a factor common to the different avitaminoses and which is capable of producing myelin sheath changes in the cochlear nerve.

VESTIBULAR PHYSIOLOGIC REACTIONS

Price and Spiegel [14] experimented on cats to determine vestibulocerebral pathways.

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11. Covell, W. P.: The Peripheral Endings of the Cochlear Nerve, Ann. Otol., Rhin. & Laryng. **47**:63 (March) 1938.

12. Covell, W. P., and Noble, L.: The Significance of Myelin Sheath Degeneration for the Cochlear Nerve, Ann. Otol., Rhin. & Laryng. **46**:895 (Dec.) 1937.

13. Covell, W. P.: A Cytologic Study of the Effects of Drugs on the Cochlea, Arch. Otolaryng. **23**:633 (June) 1936.

14. Price, J. B., and Spiegel, E. A.: Vestibulocerebral Pathways, Arch. Otolaryng. **26**:658 (Dec.) 1937.

The conduction of labyrinthine impulses to the higher centers was studied . . . by recording the cerebellar and cerebral cortical action potentials after stimulation of the labyrinth by rotation.

A special apparatus was built permitting artificial respiration and the connection of the electrodes with the recording galvanometer to be retained during rotation.

Stimulation of the labyrinth by rotation induces a functional change in the cerebellar cortex, as shown by an increase of the amplitude and number of the fast oscillations led off from the cortex of the vermis cerebelli.

Changes in the electrocorticogram similar to those induced by stimulation of other sensory nerves (increase of the fast oscillations, inhibition of the slow waves) may be elicited by the labyrinthine impulses even if the cerebellum is extirpated, and if this operation is combined with severance of the posterior longitudinal fasciculus in the anterior third of the pons.

It is inferred that the labyrinthine impulses may reach the cerebral cortex not only by way of the cerebellum-nucleus ruber system and by connections of the posterior longitudinal fasciculus with the nucleus ruber but also by pathways outside these systems. Vestibuloreticulothalamic pathways and connections between the vestibular and the cochlear tracts come into question.

Human subjects were rotated in a chair and their reaction time to motion recorded by Barter and Travis.[15] "The average vestibular reaction time to passive bodily motion was 0.598 second ranging from 0.190 to 1.79 second."

Buchanan [16] studied the value of vestibular tests in the diagnosis of central lesions in the nervous system by creating definite lesions in the brain stem and cerebellum in guinea pigs and then testing their ocular reactions. His conclusions are as follows:

The position of the eyes, the occurrence of spontaneous nystagmus and the results of caloric and rotatory stimulation of the ears in a series of 32 guinea pigs with lesions in the cerebellum and brain stem has led to the conclusion that such examinations and tests give results which are indicative of the location of the lesions.

Deviations of the eyes, apparently due to the asynergia of the eye muscles, were certainly, in the guinea pig, suggestive of the presence of the lesions and in many cases were of localizing value.

Spontaneous nystagmus which persisted for four or five days suggested brain stem involvement, and in these animals with brain stem lesions was directed toward the side opposite the lesion in the great majority of cases.

Perversion or inversion of the caloric nystagmus was certainly significant from the standpoint of incidence and, in the majority of cases, occurred only when the ear on the side of the lesion was stimulated. Changes in direction of postrotatory nystagmus occurred more infrequently, but they had a greater incidence in those animals with lesions limited to the brain stem than in those with cerebellar involvement.

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15. Barter, B., and Travis, R. C.: The Reaction Time to Vestibular Stimuli, J. Exper. Psychol. **22**:277 (March) 1938.

16. Buchanan, A. R.: Eye Deviation and Nystagmus in Guinea Pigs with Lesions of the Cerebellum and Brain Stem, Laryngoscope **47**:874 (Dec.) 1937.

Temporary adjustment of the eyes in the sense of the slow component of the nystagmus following rotation happened often enough to be of significance and usually followed rotation to the left, the lesions being placed on the right side.

Correlation of the manifestations of pathology with specific destruction of anatomical structures was not always possible since two animals with apparently identical lesions may show slightly or considerably different symptoms. This may be due to anatomical variations, but is more probably due to variations in the degree of extension of the lesions to contiguous structures in the different animals. Such variations undoubtedly also exist in the patients which have been reported in the clinical literature and account for the diversity of the results obtained from the vestibular tests.

Further, Buchanan [17] found that

Post-rotatory nystagmus is still elicitable in guinea pigs with the cerebral hemispheres and thalamus removed. Neither the slow nor the quick components of the nystagmus were disturbed by decerebration at a level which passed through the superior colliculi dorsally and the mamillary body ventrally. The direction and intensity of the nystagmus remained within normal limits.

Lesions in the right half of the cerebellum did not modify the post-rotatory nystagmus, either alone or in conjunction with decerebration.

Northington and Barrera [18] used Macacus rhesus monkeys to test vestibular reactions. The reactions obtained in the monkeys by rotation and by caloric and galvanic stimulation were tabulated and then correlated with those obtained in human beings. The authors conclude:

1. The caloric test should be used routinely . . . to determine unilateral vestibular loss of function, because the reactions to the rotation test are not dependable. The reactions, however, to the rotation test *for a variable short time* after a total unilateral vestibular loss may suggest impairment of function on the side of the lesion.

2. The galvanic test, with the electrodes placed in the hand and in the ear, should be used when there is no response to the caloric test. Normal reactions to the galvanic current indicate that the VIIIth nerve has not degenerated.

3. The direction (quick component) of the nystagmus commonly is towards the side of the normal ear in the presence of vestibular disturbances, due to an ear infection or trauma, and may be due to an irritative lesion as well as to a destructive lesion of the inner ear. The knowing of the cochlear and vestibular function present from the reactions to testing in patients with manifest disturbances of vestibular function from infection or trauma is of greater diagnostic value than is the direction of the spontaneous nystagmus, vertigo and pastpointing.

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17. Buchanan, A. R.: Post-Rotatory Nystagmus Following Decerebration in Guinea Pigs with Unilateral Cerebellar Lesions, Proc. Soc. Exper. Biol. & Med. **37**:713 (Jan.) 1938.

18. Northington, P., and Barrera, S. E.: Induced Nystagmus in Monkeys Following Peripheral Vestibular Lesions, with Clinical Correlations, Laryngoscope **47**:729 (Oct.) 1937.

Ferraro and Barrera [19] were able to diagnose clinically a pure cerebellar or a pure vestibular lesion in their controlled experiments on 300 monkeys. Many of their monkeys showed symptoms of combined lesions.

Liebetrau [20] asserts that pneumatization of the temporal bone has an effect on the latent period in the caloric test. The denser the bone, the shorter the period of latency. If the right and the left ear differ in duration of latency, a difference in pneumatization should be taken into consideration.

A curious finding was investigated by Pieré.[21] He reports on auricular nystagmus, the jerky movements of the external ear after rotation. He found it in 1 of 40 subjects. He speculates on the possible purpose of such a reaction in animals and on a possible connection between the vestibular and the facial nerve.

MÉNIÈRE'S SYNDROME

It is doubtful whether Ménière's syndrome can be considered a purely clinical, anatomic and physiologic phenomenon or whether it represents a pathologic condition. In 1861 Ménière first described a case of a well known group of symptoms produced by a lesion of the peripheral vestibular nerve. The autopsy left some doubts as to whether the condition was due to hemorrhage or labyrinthitis. Because this group of symptoms results from various causes, with exacerbations and complete remissions, Ménière's syndrome, or symptom complex, seems a better term.

In many cases the cause of Ménière's symptom complex is unknown. It is probably correct to assume that there is no one primary cause. One school has felt that this syndrome represents bacterial intoxication of the labyrinth, really focal labyrinthitis. Wright [22] is a believer of this. The summary at the close of his paper states:

> As a result of the critical analysis of seventy-three cases, the author regards the majority of cases of aural vertigo in which middle-ear suppuration is not present as being due to a chronic labyrinthitis.

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19. Ferraro, A., and Barrera, S. E.: Differential Features of "Cerebellar" and "Vestibular" Phenomena in Macacus Rhesus: Preliminary Report Based on Three Hundred Monkeys, Arch. Neurol. & Psychiat. **39**:902 (May) 1938.

20. Liebetrau, C.: Variability of Latency in Caloric Tests Affected by Pneumatization of the Temporal Bone, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **30**:261, 1938.

21. Pieré, P. F.: Systematic Investigation of the So-Called Auricular Nystagmus in Man, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **29**:597, 1938.

22. Wright, A. J.: Aural Vertigo: A Clinical Study, J. Laryng. & Otol. **53**:97 (Feb.) 1938.

As a result of his research, he believes that the condition is not secondary to middle-ear disease. From the evidence, he deduces that the labyrinthine disease results from a focus of infection, usually situated in the nose, throat or mouth.

He suggests the name of "Focal Labyrinthitis" for this disease.

A second school is represented by Hallpike and Cairns,[23] who describe

the histological changes in the temporal bones of two . . . patients [with Ménière's syndrome]. In each of these, the affected temporal bone showed a gross distension of the endolymph system together with degenerative changes in the sensory elements. A possible explanation of this distension is suggested by the finding in both cases of certain changes around the saccus endolymphaticus. . . .

The suggestion is therefore made that the endolymphatic dilatation in these cases is due, at any rate in part, to some failure in the normal absorptive mechanism; and that morphological evidence of this failure is provided by the absence from these cases of the normal absorption area of perisaccular connective tissue.

Again the site of the lesion is thought by many to be in the trunk of the eighth nerve and not in the labyrinth. This view is believed correct by Dandy, who has now operated on more than 250 patients, severing either the vestibular portion or the entire eighth nerve. His results are excellent, and he has had no fatalities.

Crowe [24] ascribes Ménière's syndrome to a disorder of the normal conditions of pressure and chemical constitution of the endolymph. His "report is based on a study of 117 patients seen . . . during the past nine years." Ninety-four of these were operated on, 49 by division of the whole auditory nerve and 45 by division of the vestibular nerve alone. The author firmly believes that the vertigo and deafness of Ménière's disease are always due to stimulation of, and degenerative changes in, the peripheral auditory and vestibular structures, that it is impossible to have vertigo after section of the vestibular nerve and that the cure of the disease is due to this fact. If this is true, one must conclude that functional vestibular phenomena are not always reliable or accurate diagnostic guides.

The strongest reason for [the author's belief] . . . that Ménière's disease is the result of pressure or chemical changes in the endolymph [which may irritate or injure the cells of the organ of Corti and have a cumulative irritative effect on the end organs of the vestibular nerve], is the frequency and length of the remissions [that are so commonly met with]. Anatomically the endolymph, and possibly the perilymph, are the only common factors in which a disorder, or one lesion, could affect both the organ of Corti and the vestibular end organs.

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23. Hallpike, C. S., and Cairns, H.: Observations on the Pathology of Ménière's Syndrome, J. Laryng. & Otol. **53**:625 (Oct.) 1938.

24. Crowe, S. J.: Ménière's Disease: A Study Based on Examinations Made Before and After Intracranial Division of the Vestibular Nerve, Medicine **17**:1 (Feb.) 1938.

With regard to other theories the author asks some pertinent questions.

> If the symptoms of Ménière's disease were due to a disorder in the metabolism of sodium or fluids, why should patients get perfectly well after division of one vestibular nerve with no change in their dietary habits?
>
> Why should the disease involve only one ear in the majority of cases?

Other theories are considered and the objections to them stated.

Furstenberg (1935) investigated the influence of the metabolism of water and salt on Ménière's syndrome. He feels that he has proved that the condition is due to retention of sodium. Mygind and Dederding,[25] on the other hand, are equally positive that retention of water is the explanation. The concluding part of their paper is to be published in the March 1939 issue of the *Annals of Otology, Rhinology and Laryngology*. They correlate clinical, anatomic and physiologic data in a simple system of explanation applicable not only to the ear but also to other parts of the body. The essential part of the mechanism of the origin of Ménière's syndrome, they feel, is a vasomotor vascular deficiency leading to local or more or less universal intracellular edema.

> The reason why these processes are clinically much more conspicuous in the ear than elsewhere in the body, is that the labyrinth is a sensory organ with a double and very sensitive function, in which the sensory and epithelial cells have a delicate osmotic regulation, necessary for the maintenance of their function in the easily influenced surrounding fluid. The slightest disturbance of this regulation leads to a swelling of the sensory epithelium resulting in an increase of the content of the labyrinth and of the endolabyrinthine pressure. . . . In the closed unyielding, hydrostatic system of the labyrinth this increase of pressure immediately elicits acoustic and very often vestibular phenomena.

The general treatment described by Mygind and Dederding consists principally in dehydration and in stimulation of vasomotor function throughout the body. The diet is low in salt but not salt free. Locally, the eustachian tube is daily catheterized, and disease in the nose, throat and tonsils is cleared up whenever present.

Two other methods of treatment of Ménière's syndrome deserve mention. Putnam [26] describes an operation which

> consists in destruction of the vestibular ganglion through an opening into the superior [semicircular] canal from the upper anterior surface of the petrous ridge, approached by a small subtemporal decompression.

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25. Mygind, S. H., and Dederding, D.: Ménière's Disease as an Indicator of Disturbances in the Water Metabolism, Capillary Function, and Body Condition, Ann. Otol., Rhin. & Laryng. **47**:55 (March) 1938; Clinical Experiments with Reference to the Influence of Water Metabolism on the Ear, ibid. **47**:360 (June) 1938; Diagnosis and Treatment of Ménière's Disease, ibid. **47**:768 (Sept.) 1938.

26. Putnam, T. J.: Treatment of Recurrent Vertigo (Ménière's Syndrome) by Subtemporal Destruction of the Labyrinth, Arch. Otolaryng. **27**:161 (Feb.) 1938.

Five operations in 2 cases are reported; in each case the vertigo was relieved while hearing was preserved. In 1 of these there was no evidence that an infection of the ear had ever existed; in the other, there had been an infection on one side but both sides had to be operated on before relief was obtained.

Peacock [27] injected alcohol into the labyrinth through the oval window of a cadaver and found that it resulted "in total destruction of labyrinthine function. The incidence of the known possible complications of facial paralysis and otitis media is thought to have been reduced to a minimum." The author has treated 2 women by this method and entirely relieved them of aural vertigo. One of these has, however recently reported some return of giddiness.

EXPERIMENTAL VESTIBULAR PHYSIOLOGY

Werner [28] asserts that endolymphatic movement usually starts in the ampulla. He obviously refers to the movement of the endolymph caused by rotation, the inertia of the lymph giving rise to a movement relative to that of the rest of the labyrinth. He bases his conclusion on his work in comparative anatomy and physiology. He has never claimed that the semicircular canals proper were of no importance in endolymphatic movement, but he thinks that the end organ of the ampulla can be stimulated without a preceding movement of the endolymph in the canal. In his opinion, the canal merely allows the endolymph to move, and by virtue of its narrow lumen it retards the flow, thus protecting the end organ of the ampulla against too violent endolymphatic movements. In animals that have been centrifuged, the cristae and cupolas remain intact.

Hasegawa [29] studied the statokinetic function of the saccule. In frogs the saccular nerves were torn on both sides, the resulting lesions being checked microscopically. The animals failed to react to movements induced along the dorsoventral axis. It is therefore the function of the saccular apparatus to register such movements. These experiments substantiated the author's previous experiments on guinea pigs.

Retjö [30] feels that sound waves stimulate the static apparatus. He refers to the investigations of Hogyes, Tullio and Bekesy and concludes

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27. Peacock, R.: Alcoholic Labyrinthine Injection Through the Oval Window in the Treatment of Aural Vertigo, Lancet **1**:421 (Feb. 19) 1938.

28. Werner, C. F.: The Anatomical and Functional Relationship Between Ampulla and Semicircular Canal: A Reply to Critics of the Author's "Ampullary Theory," abstracted, Zentralbl. f. Hals-, Nasen. u. Ohrenh. **29**:427, 1937.

29. Hasegawa, T.: The Stato-Kinetic Function of the Saccule, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **30**:364, 1938.

30. Retjö, A.: Do Sound Waves Stimulate the Static Apparatus? abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **30**:261, 1938.

that the vestibular portion of the labyrinth can be stimulated by loud sounds. This, however, elicits not acoustic sensations but muscular reflexes.

PHYSIOLOGY OF HEARING

The Middle Ear.—Experimental work on sound conduction in the auditory apparatus is reported by Kobrak,[31] who states:

> Among all experimental observations the diminution of the vibrations of the ossicles, due to muscular contraction, is the most outstanding finding. In addition, the presence of the reflex contractions during strong stimuli, and their absence in weak tones, point toward a protective function. . . .
>
> The acoustic stimulus controls the strength of the muscle contraction very accurately. Threshold observations and overthreshold recordings revealed that each acoustic stimulus has a certain constant and regular strength of contraction. The muscle contraction in turn changes the sensitivity of the ossicular chain. Each tone has therefore its own characteristic sensitivity of sound conduction. From . . . experiments the ear can be described as a sound recording device with changing sensitivity. The conditions of sound conduction are not constant. Each tone travels over an ossicular chain, the sensitivity of which is specially adapted to the physical qualities of the tone.
>
> . . . The sensitivity of the ossicular chain is increased in the resonance zone. Weak stimuli which do not produce muscle contractions utilize the over-recording. They are actually amplified. Underthreshold stimuli may be made audible. The sensitivity of the ear in the most important tone range is therefore enhanced for soft tones. Loud tones which need no amplification are cut down by muscle reflex.
>
> . . . The middle ear has a resonance frequency located exactly in the area of the spoken voice.

Wever and Bray[32] used the electrical responses of the cochlea to determine the function of the tensor tympani muscle. They report:

> The responses to various tonal stimuli were measured under normal conditions and during the application of artificial tension to the tensor tympani tendon.
>
> . . . [They found the] functional relations of tension and stimulus frequency [to be] . . . complex. [Their] . . . results . . . suggest that in addition to its reduction of sound conduction, tension raises the natural frequency of the transmission system of the ear. These two effects combined give a relation to frequency which is determined by the particular form of the sensitivity curve of the ear.

In contrast, Mink[33] practically discards the ossicles in his conception of sound conduction. He maintains that

> the molecular movement of sound is conducted by way of the drumhead and the air in the tympanic cavity to the inner ear. The lateral wall of the labyrinth

31. Kobrak, H. G.: Experiments on the Conduction of Sound in the Ear, Ann. Otol., Rhin. & Laryng. **47**:166 (March) 1938.

32. Wever, E. G., and Bray, C. W.: The Tensor Tympani Muscle and Its Relation to Sound Conduction, Ann. Otol., Rhin. & Laryng. **46**:947 (Dec.) 1937.

33. Mink, P. J.: The Auditory Picture, Monatschr. f. Ohrenh. **72**:603 (June) 1938.

is affected by this movement, which spreads out in the labyrinth capsule. From the promontory, the sound apparently reaches the lamina spiralis ossea, the membrane of Corti and the hair cells. The latter receive all tones of the same pitch—contrary to the Helmholtz theory of the simultaneous reception of multiple tones. If one judges from the findings in lower animals, there is a center in the recessus sphaericus vestibuli which retransmits the vibrations so that they stimulate the hair cells of the macula sacculi. Similarly, there are probably two centers in the recessus ellipticus vestibuli which, besides stimulating the macula utriculi, have close connections with the semicircular canals.

Three centers are therefore concerned in the interpretation of the component parts (*Teiltönen*) of a sound. Together they build up a sensation of the sound's characteristics in the conscious mind and also influence equilibrium. In this sensation lies the recognition of pitch, to which the cochlea adds the intensity of the sound and so completes the auditory picture.

Bone Conduction.—The physiology of bone conduction is described by Kulikowsky.[34] In order to study the resonating properties of the human head, tuning forks were applied to macerated skulls and the vibrations of the latter examined by means of the Simon oscillograph. It was found that the skull is a complex resonator and that it responds only to tones of low and medium pitch, reacting best at 160 double vibrations. When the skull was filled with wax or petrolatum the optimum response was at 80 double vibrations; i. e., it responded better to low tones. Studies on living subjects, in normal and in pathologic states, yielded the following results: 1. An exact measurement of bone conduction can be done only in a sound-proof room, called by the author the "deaf chamber." 2. In the "deaf chamber" a low fork was heard for two hundred and thirty seconds by bone conduction, whereas in any ordinary room it was heard only for ninety seconds. For higher tones the difference was less. 3. Patients with disease of the middle ear did not show increased bone conduction in the "deaf chamber." On the contrary, bone conduction was always shortened, although not as much as with labyrinthine disorders.

The Cochlea.—Pohlman[35] considers the objections to the accepted interpretation of cochlear mechanics. He says:

E. H. Weber correctly assumed the middle ear apparatus to have the function of an impedance matching transformer which helped to balance out the resistance differences between the air and the labyrinth liquid. However he disregarded this interpretation in his further analysis of cochlear response. He believed because the labyrinth liquid was quite incompressible and the otic capsule was relatively inelastic that a compensation area must be present to permit the labyrinth liquid to "accept" the vibrations of the stapes. The result of this scheme was the

34. Kulikowsky, G. G.: The Physiology of Bone Conduction, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **29**:288, 1937.

35. Pohlman, A. G.: Objections to the Accepted Interpretation of Cochlear Mechanics, Acta oto-laryng. **26**:162, 1938.

translation of the longitudinal displacements in the scalae into transverse vibrations in the basilar membrane. This interpretation forms the basis for all theories on indirect activation . . . [but the] shock-absorbing function of the internal ear was mistaken for the displacements in the liquid essential to audition.

According to Hallpike and Rawdon-Smith,[36] the discoverers of the "Wever and Bray Effect"

showed that, when electrodes are placed on the eighth nerve of a cat, alternating potentials synchronous with an acoustic stimulus applied to the ear may be readily elicited. Wever and Bray's original view as to the nature and origin of this phenomenon, namely, that the potentials were due to the passage of impulses in the eighth nerve, is no longer held adequate; . . . the effect is of a dual origin —in small part due to the passage of impulses, but in greater due to the electrical spread of alternating potentials generated within the cochlea. . . . various theories . . . propounded as to the origin of this latter component . . . may be conveniently divided into two groups, which have been termed neural and non-neural. Critical examination leads to the conclusion that, of all these, the most probable is the so-called "Membrane Hypothesis." On this view the potentials are held to arise owing to movements of a polarized membrane, probably the membrane of Reissner.

Adrian [37] agrees that "The most plausible explanation of the effect is that of Hallpike and Rawdon Smith, namely, that it is due to the vibration of a polarized membrane separating endolymph and perilymph." Adrian points out that there is a "similar effect in the inner ears of cold-blooded vertebrates," alligators, lizards, tortoises, eels and pike.

The frequency of the potential changes which can be induced in the inner ear seems to be limited merely by the frequency at which the structures can be made to vibrate; the maximum frequency of response in the fibres of the auditory nerve is much lower, since there must be time for recovery after each impulse.

Kupfer [38] demonstrates that

the electro-acoustic effect and the origin of the Wever-Bray response are linked in fact and mathematics. . . . [He explains] The *electro-acoustic effect* and the *Wever-Bray phenomenon of the ear* . . . by the disturbance and displacement of Freundlich's electrical double layers at phase boundaries of liquids and solids in the cochlea. These double layers of potential ζ may be displaced by movements of the stapes as well as by an external E.M.F. Tinnitus being understood as an electrical phenomenon [due to currents in a diseased organ can be] . . . neutralized in many cases by . . . *electrotherapy*.

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36. Hallpike, C. S., and Rawdon-Smith, A. F.: The Wever-Bray Phenomenon: A Summary of the Data Concerning the Origin of the Cochlear Effect, Ann. Otol., Rhin. & Laryng. **46**:976 (Dec.) 1937.

37. Adrian, E. D.: Potential Changes in Ears of Reptiles and Fish, Nature, London **141**:553 (March 26) 1938.

38. Kupfer, E.: On the Origin of the Wever-Bray Response and on an Electrotherapy of the Ear, J. Laryng. & Otol. **53**:16 (Jan.) 1938.

This therapy must be repeated often and "requires a . . . considerable amount of patience and care."

These views are contradicted by Davis and Lurie and their co-workers,[39] who claim that the cochlear electrical response is due to the function of the organ of Corti itself. In experiments with guinea pigs, areas of the organ of Corti were destroyed mechanically or by drugs, and it was observed that the electrical response decreased in proportion to the amount of damage in the hair cells.

In congenitally deaf guinea pigs with a normal Reissner's and basement membrane but with absence of the organ of Corti in the lower, or first, three of the four turns, cochlear response was absent for these regions. But when there was a functioning organ of Corti at the helicotrema, cochlear response for low tones was obtained by intense stimulation.

This experimental evidence indicates that the origin of the Wever and Bray phenomenon cannot be in Reissner's membrane. It shows that it must be in the hair cells of the organ of Corti and in the nerve endings. Wever and Bray [40] state:

> No absolute threshold has been discovered for the cochlear response, and there is no reason to suppose that any exists. The origin of the Weber relationship between stimulus intensity and loudness must be sought in processes central to the receptor. The character of the functional relationship between loudness and frequency also is probably dependent upon central processes, though it doubtless reflects in an indirect manner the properties of the end-organ.

Bordley and Hardy [41] find

> little room for doubt that the Wever-Bray phenomenon is closely related to hearing acuity in animals. . . . The effect of experimental incisions of the tympanic membranes on cochlear potentials in cats is compared to the effect on the hearing acuity in man of uncomplicated traumatic rupture of the tympanic membrane. The two are similar, at least for the octave frequencies from 256 to 4096 double vibrations per second. This [experimental] evidence supports the view that changes in cochlear potentials (Wever-Bray phenomenon) parallel changes in hearing acuity and therefore that experimental observations with this technic do provide information that can properly be applied to the interpretation of clinical tests of hearing.

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39. Lurie, M. H.: Symposium—the Neural Mechanism of Hearing: Pathology of the Organ of Corti, Laryngoscope **47**:418 (June) 1937. Davis, H.; Dworkin, S.; Lurie, M. H., and Katzman, J.: Symposium—The Neural Mechanism of Hearing: Animal Investigations; Behavioral, Electrical and Anatomical Studies of Abnormal Ears, ibid. **47**:435 (July) 1937.

40. Wever, E. G., and Bray, C. W.: Nature of Acoustic Response: Relation Between Stimulus Intensity and Magnitude of Cochlear Responses in the Cat, J. Exper. Psychol. **22**:1 (Jan.) 1938.

41. Bordley, J. E., and Hardy, M.: Effect of Lesions of the Tympanic Membrane on the Hearing Acuity, Arch. Otolaryng. **26**:649 (Dec.) 1937.

In conclusion, recent advances in knowledge of sound conduction are practically applied to cases of conduction deafness by Pohlman.[42] He states:

First, . . . as the drum membrane and ossicular chain in the human being is important in the effective conduction of air sounds of all frequency to the cochlea, then where the drum membrane shows . . . a wide dehiscence, it may be patched.

Second, where the sound conduction apparatus has been removed, it may be replaced by an efficient prosthesis provided that the external auditory canal is sufficiently straight and wide and the stapes area is movable.

Third, where the middle ear apparatus is functionally out of commission by reason of adhesions in the middle ear, or perhaps a chronic otitis media which does not yield to treatment, a similar removal and replacement is a possibility. The drum membrane may be reflected and the use of a simple acoustic probe will reveal whether or not the stapes area is sufficiently movable. . . . The probe consists of a half circle diaphragm of mica supporting a bamboo stem or a wire. The application is simple and devoid of any danger, and the voice of the operator projected against the mica diaphragm will give all of the information required.

Hughson[43] adds:

1. Impaired bone conduction is not necessarily an indication of atrophy of the nerve or organ of Corti.

2. Loudness balances and fatigue tests are valuable adjuncts in differentiating between nerve and conductive deafness.

3. Toxic neuritis of the cochlear division of the eighth nerve undoubtedly plays an important role in the development of certain cases of deafness.

4. Accurate determination of the etiologic factor is usually possible and appropriate therapy will arrest the progress of the impairment [in hearing] and in many instances bring about improvement.

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42. Pohlman, A. G.: Recent Contributions to Our Knowledge of the Auditory Apparatus and Their Relations to Disabilities in Hearing, Tr. Am. Laryng., Rhin. & Otol. Soc. **43**:257, 1937.

43. Hughson, W.: The Inner Ear from an Experimental and Clinical Standpoint, Ann. Otol., Rhin. & Laryng. **47**:69 (March) 1938.

Abstracts from Current Literature

Ear

THE USE OF SULFANILAMIDE IN ACUTE MASTOIDITIS: REPORT OF CASE. M. VAISBERG, Laryngoscope **48**:54 (Jan.) 1938.

Vaisberg presents a case of acute perforative zygomatic mastoiditis probably due to a hemolytic streptococcus, with a secondary cardiac complication. The intensive use of sulfanilamide combined with some antistreptococcus serum was effective in rapidly overcoming the mastoiditis and the apparent secondary cardiac complication. A total of 1,200 grains (78 Gm.) of sulfanilamide was given in twenty-eight days to a child aged 12 years. This averaged 42.9 grains (2.78 Gm.) daily. The only evident disturbances which might have been due to the drug were the cardiac manifestations and the pains in the joints, but these came on during a period when the doses were much reduced. The remarkably rapid diminution of perizygomatic swelling and the rapid flattening of the drum after the institution of sulfanilamide therapy changed the picture from one of acute surgical mastoiditis to one of rapidly resolving acute otitis media. The part played by the antistreptococcus serum is difficult to evaluate, but it seems that the sulfanilamide, which was used for both the mastoid and the heart successfully, was the deciding factor in the favorable outcome. Since this case was observed, many patients with proved streptococcic infections of the middle ear, throat and larynx have been treated intensively with sulfanilamide with no apparent immediate ill effects other than moderate cyanosis. In 1 case there were a papulomacular rash and some nausea, which disappeared within a few hours after the drug was discontinued.

EDITOR'S ABSTRACT.

POLYMORPHISM OF TUBERCULOSIS OF THE EAR. J. TERRACOL and DE GANDIN DE LAGRANGE, Rev. de laryng. **59**:254 (March) 1938.

"The rarity of auricular tuberculosis is a clinical illusion."—Lermoyez. Tuberculosis of the ear is an "open" lesion; hence a secondary infection is present. The lung is not necessarily involved. The aural infection may be accompanied by profuse discharge, development of carious bone, exposure of the carotid artery and meningitis. The 4 cases reported indicate the polymorphism of tuberculosis of the petrous pyramid.

CASE 1.—A child of 3½ years who showed "white caries" of bone on operation died of septicemia. Inoculation of the pus from the ear into a guinea pig proved the condition to be tuberculous.

CASE 2.—A Senegalese rifleman had parotid pain and an abscess with grumous discharge. Removal of aural polyps was followed by a "cold" sternal abscess. The patient's cutaneous reaction was positive for tuberculosis. Laboratory findings otherwise were negative. Headache and rigidity of the neck developed, and mastoidectomy was performed. The carotid area was involved. One month later the patient died of fulminating hemorrhage from the carotid, but autopsy showed numerous tuberculous lesions. The diagnosis was primary tuberculosis of the temporal bone with secondary somatic invasion.

CASE 3.—A child of 2 years had bilateral aural infection. The Koch bacillus was found on direct examination of the pus from the mastoid cavity. Histologic tuberculosis was seen in the granulations. A guinea pig inoculated with the material showed a positive reaction. The child had a concurrent pulmonary lesion. One year later he appeared to be in good health.

CASE 4.—A man aged 37 had a polyp in the right canal which was histologically tuberculous and a lesion in the labyrinth. Thoracic examination showed bilateral tuberculosis. The biopsy permitted the diagnosis of tuberculous involvement of the ear.

There are two clinical forms of otitic tuberculosis, the primary and the secondary. Each form may be acute or latent. The acute type, the rare type of Schwartze, begins with an intense pain, and there may be much discharge. The latent type of Schweibe, is more frequent. The otitis begins insidiously. Loss of hearing and multiple perforations occur. Complications in the primary form depend on the extent of the bone lesions and include paralysis of the facial nerve, mastoiditis and labyrinthitis. Repeated hemorrhages and fatal hemorrhages due to erosion of the carotid canal are not exceptional.

Classified anatomically there are five forms:

(*a*) Progressive infiltration.
(*b*) Necrosis with sequestration.
(*c*) Chronic osteitis of the middle ear with hyperostosis of the mastoid.
(*d*) Rarefaction and ulceration.
(*e*) Osteitis of the external canal or of the squamous part of the bone.

Inoculation of a guinea pig is a great aid in making the diagnosis. Obviously, many times the diagnosis is made postoperatively. The authors feel that here is one situation in which the old adage "Tuberculosis does not like the knife" should be revised. However, the classic treatment, especially postoperative heliotherapy, must be faithfully followed.

BATSON, Philadelphia.

SUBCONSCIOUS AUDITORY PHENOMENA (SUBJECTIVE PERCEPTION OF SOUND): ITS ROLE IN DYSACOUSIS. E. ESCAT, Rev. de laryng. **59**:321 (April) 1938.

Only physioclinical experiments permit the study of subconscious auditory phenomena. Physioclinical methods applied to man and experimental methods used for animals are complementary and not contradictory. It is impossible to experiment with animals on the subjective auditory phenomena of man. For example, the existence of tinnitus is known solely from the history of the patient. The physioclinical observations are particularly important when the patient has some knowledge of sound or music. Much can be learned by observation of one's own ears, whether normal or showing dysacousis. Patients with disease of the ear but no discharge are divided into those who complain of subjective sensations of hearing and those who do not. The sensations may be continuous or intermittent. The author cites the complaint of an otosclerotic patient that the tinnitus masks his hearing. The acuity of hearing varies inversely with the tinnitus. Typical loss of hearing in otosclerosis is for low tones, while the typical note of tinnitus is high pitched. The author believes that patients who say that they have no tinnitus actually have subconscious tinnitus.

Conclusions.—If one admits that an objective sound may be masked by a more intense subjective tone, the author believes, dysacousis may result from similar masking by a subconscious subjective tone. He believes this is one of the phenomena by which a hiatus is produced and has endeavored to demonstrate that the lacunas in the auditory spectrum are assignable to absorption rather than to actual deficiency in certain zones of the acoustic scale.

BATSON, Philadelphia.

PETROSQUAMOUS OSTEOMYELITIS IN INFANTS. A. LASKIEWICZ, Rev. de laryng. **59**:342 (April) 1938.

Three cases of children with involvement of the petrous apex are discussed, together, with 2 cases of adults. One child came to autopsy. Infected thrombi in the haversian canals were noted microscopically. The observations tended to

confirm the present opinion that infection is propagated by way of the vascular system. The author reviews extensively the various current classifications of mastoid cells. The prominent clinical symptoms of osteomyelitis of the petrous apex are: parietal headache of the involved side; pain in the occiput and in the orbit; involvement of the fifth, the sixth and more rarely of the seventh, the eighth, the ninth and the tenth cranial nerve, and commonly edema in the zygomatic region, which may extend to the lids and the occiput. Serious attacks lead to meningitis. Progression seems to be along the carotid canal.

BATSON, Philadelphia.

IMPAIRED HEARING FOR HIGH TONES. SAMUEL J. CROWE and STACY R. GUILD, Acta oto-laryng. **26**:138, 1938.

The authors call attention to the classic teaching that lesions of the middle ear cause more impairment of hearing for low tones than for high tones and that when there is an impairment for high tones in the presence of a lesion of the middle ear the diagnosis of "combined deafness" should be made. Crowe and Guild have long believed that this clinical picture can be produced by lesions of the middle ear alone, and this belief was based on the fact that in several cases with this diagnosis the inner ear was histologically normal. Three years ago, with Polvogt, they studied histologically 79 ears of adults with a "gradual high tone loss," and since then they have studied the sections in 40 additional cases. The typical lesion in these ears with "gradual high tone loss" is partial atrophy of the cochlear nerve in the osseous spiral lamina of the basal turn. The organ of Corti and the external sulcus cells were normal. In cases of an "abrupt high tone loss" (good hearing for tones up to 1024 or 2048 double vibrations and a drop of 40 decibels or more in the next succeeding octave) the lesion consists of total atrophy of Corti's organ and the external sulcus cells in some portions of the basal turn, together with atrophy in the cochlear nerve.

The authors believe that the "combined type" of deafness is in some cases caused by occlusion of the orifice of the eustachian tube by lymphoid tissue, and they report 25 such cases, 1 in extenso, in which they have treated the lymphoid tissue about the orifice of the tube by roentgen rays, radium or a combination of both. In some of these cases there has been marked improvement in the hearing for all tones including the high tones and a switch in the result of the Rinne test from negative to positive. Their best results in this group of 25 cases have been with children under the age of puberty. They use no more than 2.2 gram minutes of radium emanation in a gold applicator placed next to the orifice of the eustachian tube under the direct vision of a nasopharyngoscope introduced through the other side of the nose. The application is made in this way to avoid any stimulation or irritation of the hypophysis. They have observed no unfavorable results from this treatment.

GROVE, Milwaukee.

Pharynx

MEDICAL COMPLICATIONS OF TONSILLAR INFECTIONS. R. DUPERIE, Rev. de laryng. **59**:333 (April) 1938.

1. The tonsil may act as a portal for entry of contagious diseases and infections.

2. The tonsil plays a role as a site of focal infection.

3. The tonsil may give rise to septicemia due to either aerobic or anaerobic organisms.

The author decries surgical intervention, including both tonsillectomy and jugular ligation, either early or late, for acute tonsillar infections. Medical treatment consists of the use of antistreptococcus serum and of derivatives of acridine. Certain organisms have no specific antiserums. Treatment is symptomatic, with evacuation of collections of pus as they localize.

BATSON, Philadelphia.

INOPERABLE SARCOMA UNTREATED FOR FOUR YEARS HEALED BY ROENTGEN THERAPY. T. SCICLOUNOFF, Rev. de laryng. **59**:392 (April) 1938.

The experience with 33 malignant tumors of the tonsillar area is recounted. One of these, unsuited for operation, was seen at the clinic four years after the onset of symptoms. The patient had received no previous treatment. After roentgen therapy he has been free of symptoms for four years at the time of the report.

BATSON, Philadelphia.

Larynx

SOME CLINICAL ASPECTS OF INACTION OF THE VOCAL CORD. HERBERT TILLEY, J. Laryng. & Otol. **53**:353 (June) 1938.

Laryngeal paralysis in vocal cords which appear to be normal and whose condition cannot be ascribed to a gross lesion involving the recurrent laryngeal nerve is considered.

Eighteen cases are presented. In 6 temporary aphonia with paralysis of the cord lasting from a few days to six months resulted during the course of acute infection of the upper part of the respiratory tract, supposedly from peripheral neuritis of toxic origin. Permanent fixation of the cord, due presumably to cricoarytenoid ankylosis, was observed in 2 cases following severe streptococcic laryngitis. In 1 case of severe hemolytic streptococcic laryngitis, tracheotomy was required to relieve the dyspnea consequent on the edematous reaction, but no permanent paralysis ensued. In 1 case acute laryngitis with temporary paralysis was caused by acute rheumatic fever. In 5 cases hoarseness with paralysis of the cord developed in apparently healthy persons. In all the cases the symptoms disappeared without treatment, but the condition was never explained.

Aortic aneurysm was responsible in 1 case, and in another paralysis of the cord occurred in a tuberculous patient who exhibited no signs of laryngeal phthisis. In the latter, it is admitted, the recurrent laryngeal nerve may have been directly involved in a pleuritic adhesion. In 1 case temporary paralysis occurred as the result of vocal strain.

It was formerly considered that paralysis of the recurrent laryngeal nerve was a serious condition, as it was thought to be almost always an expression of some intracranial involvement. While it often is, there are many instances in which the cause is of a comparatively minor nature.

Of great interest in the review of these cases is the frequency with which the left side of the larynx was involved. It was noted that in mild acute laryngitis edematous reaction was limited to the left side. Acute hemolytic streptococcic laryngitis causing bilaterally symmetric edema resulted in permanent paralysis on the left side and recovery on the right. In the case in which the condition was due to vocal strain, only the left cord was involved. In only 1 case, one of those in which the cause was unknown, was the right cord impaired.

No satisfactory explanation for this phenomenal vulnerability of the left recurrent laryngeal nerve can be advanced. It is known that the left nerve is longer than the right and hence is more susceptible to compression by pleural and mediastinal disease; but when toxemia is responsible for neuritis the increased length should not increase the susceptibility to involvement.

LEJEUNE, New Orleans.

LARYNGOTUBERCULOSIS AND ASSOCIATED LESIONS. J. ALONSO, Rev. de laryng. **59**:472 (May) 1938.

Polyps, cysts and benign tumors of the larynx in tuberculosis are not essentially associated with the tuberculous lesion. Successful localized removal is followed by primary healing. Advance in the diagnosis and the treatment of

syphilis is rendering severe lesions of the larynx rarer. They may exist in the tuberculous, with or without a tuberculous laryngeal lesion. Scarring in the larynx is commonly due not to tuberculosis but to syphilis. The differential diagnosis must be made between syphilitic scarring and that produced by radium therapy. When the lesions of syphilis and tuberculosis coexist in the larynx, diagnosis is much more difficult. The coexistence of cancer of the larynx and tuberculosis of the larynx is relatively rare for several reasons, among which may be mentioned the difference in the ages at which the two diseases attack the patient and in the constitutional types subject to them and the relatively short course of each.

The author's statistics include data on 435 cases of epithelioma of the larynx, in 18 of which tuberculosis was coexistent. Of these 18, surgical treatment, with or without radium, was employed in 10. Two patients refused operation, and these, with the remaining 6 (a total of 8), were treated by radiotherapy.

BATSON, Philadelphia.

Miscellaneous

THE LEUKOCYTE RESPONSE TO SULFANILAMIDE THERAPY. JOHN A. BIGLER, W. M. CLIFTON and MARIE WERNER, J. A. M. A. **110**:343 (Jan. 29) 1938.

Bigler and his associates conducted a detailed study of the leukocyte response to sulfanilamide in 33 patients; some had infection and others did not. Daily leukocyte counts were made before, during and after the administration of the drug. Schilling counts, the lymphocyte-polymorphonuclear ratio and the appearance of early cells were recorded. The authors reached the following conclusions: 1. Sulfanilamide seemed to cause a depression of the white cells, even to a point at which a leukopenia developed. 2. Agranulocystosis or granulopenia did not occur with this depression, or leukopenia. 3. The action of sulfanilamide seemed to be independent of the leukocytes, in that it did not produce an increase in the total leukocytes or in the proportion of the polymorphonuclear cells. 4. Cyanosis occurred in 7 cases, but spectroscopic examination of blood specimens in these cases did not reveal bands of sulfhemoglobin or methemoglobin. 5. Sulfanilamide is a very effective drug in the treatment of infection due to beta hemolytic streptococci. 6. Frequent blood counts should accompany sulfanilamide therapy.

GORDON, Philadelphia.

CULTURE OF HUMAN MARROW: STUDIES ON THE MODE OF ACTION OF SULFANILAMIDE. EDWIN E. OSGOOD and INEZ E. BROWNLEE, J. A. M. A. **110**:349 (Jan. 29) 1938.

Osgood and his assistant undertook the study of the mode of action of sulfanilamide, using the simple method of culture of the human marrow. All the experiments were conducted with infections caused by beta hemolytic streptococci.

The methods, observations and technic were exacting and most painstaking, and the summary arrived at is as follows: The major action of the sulfanilamide on the beta hemolytic streptococcus seems to be neutralization of the toxins. Either by this means or in some unexplained manner the rate of cell division of the organisms is diminished. The drug does not directly effect phagocytosis; neither does it kill the organisms, though it permits the bactericidal properties of human serum and to some extent phagocytosis to kill them. The effective concentration of sulfanilamide appears to be 1:100,000, or only one tenth of that now ordinarily maintained in the blood stream, though this will have to be confirmed by numerous controlled experiments before it is justifiable to employ smaller doses in treating dangerously ill patients. Greater concentrations than those generally employed clinically do not appear to have direct toxic action on the nucleated cells of the majority of bloods or marrows. This applies in cases

in which there is idiosyncrasy to the drug. The effects of sulfanilamide on all other organisms should be studied and determined by the methods detailed. Cultures of human marrow should aid materially in any study of the mode of action of either a noxious or a therapeutic agent.

GORDON, Philadelphia.

USE OF IODIZED OIL IN THE VISUALIZATION OF CEREBRAL ABSCESSES OF AURAL AND NASAL ORIGIN. A. LASKIEWICZ, Rev. de laryng. **59**:217 (March) 1938.

Cerebral abscesses may result from an infection passing directly through the tegmen tympani or from retrograde thrombophlebitis. They tend to develop in the white substance of the temporal lobe, especially in its inferior angle and somewhat toward its anterior portion, and are found in the richly vascularized cortical layer only rarely, when due to osteomyelitis of the petrous pyramid, of the subarcuate fossa or of the carotid canal.

The abscesses typically have a definite capsule; the atypical form without capsule, is a localized encephalomalacia. In its first stage it may be encephalomalacia rubra due to hemorrhage. Neumann believes that the organism (for example, the diplococcus [Diplococcus crassus] and the pneumococcus) may determine the formation of a capsule. Unfavorable for the formation of a capsule are the gram-negative anaerobes, such as Bacillus coli and the staphylococci. Ghon described (in a single case) an abscess with a capsule, in which the organism was the anaerobic Bacillus sporogenes putrefaciens. Multiple abscesses of the temporal lobe, according to Fremmel and Hofmann, are really abscesses with diverticula. Large abscesses flatten the convolutions and compress the ventricle.

The abscess capsule is composed as follows: Innermost is the granular layer of granulocytic neutrophils and lymphocytes; next, a layer of necrotic cerebral tissue, and external to this, a layer of connective tissue of vascular origin, consisting of fusiform cells, round cells and cells with vesiculated nuclei and hazy outlines. The capsule is typically from 1 to 5 mm. thick, according to the age of the abscess and the type of infection. The author believes, as do others, that in cases of ventricular perforation the choroid plexus acts as a tamponade, much like the omentum in the abdomen.

The symptoms of spontaneous perforation of an abscess into a ventricle are loss of consciousness, somnulence, Cheyne-Stokes respiration, irregular and feeble pulse, sudden rise in temperature, vertical nystagmus and dilatation of the pupils. When perforation threatens, the neutrophilic granulocytes in the spinal fluid are increased, and the Pandy, Nonne-Apelt and other signs are positive. Neutrophils in the abscess itself increase with pending perforation. When the abscess perforates to the base the cardinal signs of meningitis are present: bilateral deafness, homonymous hemianopia, involvement of the oculomotor nerve and cloudy spinal fluid containing albumin and cellular elements.

To indicate the presence of an abscess fistula and to determine the dimensions and form of an abscess, Siccard, Forestier and others used contrast roentgenography. Laskiewicz introduces 2 to 3 cc. of a 40 per cent solution of iodized oil through a fine needle, after having aspirated an equal quantity of pus (method of O. Mayer). This amount of fluid, warmed to body temperature, mingles rapidly with the contents of the abscess and permits an exact depiction by roentgenography of size, diverticula and perforations. The author reports 10 cases, in 6 of which he was enabled to show diverticula of the abscess and in 3 to demonstrate the presence of a fistula. Of 5 cases reported in detail recovery occurred in 1 and death in 4, for 3 of which the complete postmorten observations are recorded. The author feels that the procedure of roentgenography with the aid of iodized oil is of considerable diagnostic importance and is an aid in planning treatment.

BATSON, Philadelphia.

FACIAL SPASM OF DENTAL AND SINAL ORIGIN. BERCHER and GUILLERMIN, Rev. d'oto-neuro-opht. **16**:183 (March) 1938.

Bercher and Guillermin discuss the pathogenesis of facial spasm on the basis of the observation of 2 cases of intractable type. Both patients recovered after the foci of irritation in the teeth and sinuses were removed. The irritation arose in the sympathetic nerve branches accompanying the alveolar or pulpal arteries, setting off a reflex which reached the trunk or branches of the homolateral facial nerve. In cases of facial spasm, it is important to remove any source of chronic irritation about the face capable of setting up motor disturbances through a chronic pathologic reflex.

DENNIS, San Diego, Calif. [ARCH. NEUROL. & PSYCHIAT.]

FOCAL INFECTION AS A PRIMARY FACTOR IN AGRANULOCYTOSIS. E. WESTERVELT DENNIS, Pract. oto-rhino-laryng. **1**:174 (July) 1938.

The author cites 4 cases of agranulocytosis observed in Syria in the last five years, in which the cause seems to have been focal infection in the mastoid, in a peritonsillar abscess and in the teeth. In 2 of the cases (mastoid and peritonsillar abscess) recovery followed surgical drainage of the infected area. In the other 2 the patient died.

The belief is stated that the syndrome of agranulocytosis may be caused by the liberation of a toxic leukocidal substance which affects the neutrophils and which if present in a sufficient quantity or for a sufficient time will injure the granulopoietic elements of the bone marrow. The author does not believe that infection of the blood stream, or septicemia, was a factor in these cases. Furthermore, there was no association in any of his cases with any drugs of the benzene ring group.

In his conclusions, he states that pyogenic bacteria from a focal infection may induce profound granulocytopenia.

PERSKY, Philadelphia.

PLETHYSMOMETRY OF NOSE FOR DETERMINATION OF PATENCY OF SIGMOID SINUS AND JUGULAR VEIN. H. A. E. VAN DISHOECK, Acta oto-laryng. **26**:43, 1938.

Stating that the Queckenstedt or Tobey-Ayer procedure is the only method at present available for determining the permeability of internal jugular veins and that a lumbar puncture is necessary to carry out the test, Dishoeck calls attention to another diagnostic test for this purpose: the observation and measurement of the intumescence of the inferior turbinates while pressure is made on the venae jugulares. By rhinoscopic examination through the anterior nares one can observe that the space between the septum and the inferior turbinate narrows as one compresses the jugular vein of the same side and widens when the pressure is released. Under such circumstances one can assume that the sigmoid sinus and jugular vein of that side are not occluded. The author has also brought out a simple but ingenious manometer, which he calls a nasal plethysmometer, for measuring changes in the erectile tissue of the nose during compression of the neck. The apparatus consists of a U tube partially filled with water, each arm of which is connected with a rubber finger cot balloon. The entire system is closed, and the balloons can be equally inflated with air to a given amount. The balloons are treated with petrolatum and introduced one into each inferior meatus. If obstruction exists in one sigmoid sinus or jugular vein, compression of the neck on that side causes no change in the level of fluid in the U tube. Compression of the other side of the neck will cause a displacement of the level. The apparatus can be used unilaterally as well as bilaterally. Dishoeck cautions that only light but firm pressure should be made on the neck. If too strong a pressure is exerted the carotid artery also will be compressed, and the anemia thus produced would defeat the test. The test can fail under the

following conditions: (*a*) if a septal crest or spur is present, (*b*) if the erectile tissue is poor because of atrophy or previous conchotomy, (*c*) if the nose is already maximally swollen by inflammation, (*d*) if because of infection in the neighborhood of the pterygomaxillary plexus of veins or by thrombosis of this plexus the connection between the veins of the turbinate and the endocranial venous system is disturbed. The response can also fail, even though the sigmoid sinus and jugular veins are patent, in the presence of an excessively large emissary vein. With these exceptions the test is a valuable one for: (1) sinus thrombosis in aural disease; (2) thrombosis of a jugular vein in infections of the neck and larynx; (3) pressure on the transverse sinus by a tumor or abscess of the brain; (4) congenital failure of the transverse sinus, the sigmoid sinus or the jugular vein.

GROVE, Milwaukee.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

THOMAS J. LEWIS, M.D., *President*

Regular Meeting, Oct. 3, 1938

WALTER H. THEOBALD, M.D., *Secretary*

POSTURAL TREATMENT IN ACUTE LARYNGOTRACHEOBRONCHITIS (Moving Pictures). Presented by DR. THOMAS C. GALLOWAY.

This paper will be published in full in *The Journal of the American Medical Association.*

DISCUSSION

DR. JOSEPH BRENNEMANN (by invitation): I appreciate the honor of being asked to discuss this paper. It deals with a subject in which I am deeply interested. The regional specialist and the general specialist naturally look at the same subject from different points of view. The great majority of papers on acute laryngotracheobronchitis have been presented by otolaryngologists and bronchoscopists. I shall approach the subject as a pediatrician. From the standpoint of pediatrics I cannot agree with the speaker that "this condition should be easy to diagnose." As a matter of fact, it often presents great difficulty in diagnosis. There are all gradations from simple catarrhal laryngitis through laryngotracheitis to laryngotracheobronchitis, and they have much in common. Only when there is laryngitis, subglottic edema and a characteristic involvement of the trachea and bronchi is one justified in calling the condition true laryngotracheobronchitis. Clinical or bronchoscopic evidence of partial or complete obstruction of some of the lower airways is commonly necessary to establish the diagnosis. Mortality statistics will vary greatly if this criterion is not rigidly adhered to.

I agree with Dr. Galloway that one should "interfere early"—but not too early. My colleagues and I have watched borderline conditions for several days and "gotten away" with it, employing only the usual palliative measures. One must not wait, however, until the child is seriously embarrassed or is getting worn out.

The chief interest in this paper centers on the value of postural drainage. It does not seem to me that the conditions present in abscess of the lung and bronchiectasis are strictly analogous to those found in laryngotracheobronchitis. The more abundant fluid and less tenacious purulent secretions of the former are quite unlike the tough, gummy, ropy, crusty masses of fibrin, desquamated epithelium and inspissated mucus that cling to the walls of the trachea and bronchi in the latter condition. I do not see how a mere 15 degree elevation of the foot of the bed will in itself lead to the removal of such masses by gravity. However, with the bed in this position it is possible to irrigate the trachea with larger amounts of fluid than we have used, and from this point of view the measure may well be a safe and useful adjunct. We have not tried it.

The final test of any method of treatment is the result. I have purposely avoided the word "statistics"—comparative statistics are worthless, even misleading, unless due attention is paid to age, type of case and treatment and the all-important adherence to rigid diagnostic criteria. It will be remembered that Baum in his large series got the best results with intubation. So did Lyman Richards. In his early reports Richards called attention to a very high mortality with tracheotomy and yet rightly, in my opinion, recommended tracheotomy as the method of choice. He questioned, and I again agree with him, whether a case

that ends favorably with intubation alone is one of true laryngotracheobronchitis. We have recently published the results we obtained over a ten year period at the Children's Memorial Hospital, where all the children were seen by one otolaryngologist. From 1927 to 1935, inclusive, we had 33 patients who were treated instrumentally, while 4 died without such treatment. Our mortality was nearly 50 per cent. In 1936 we had an adequate bronchoscopic outfit and good bronchoscopists, easily accessible. The 12 children encountered during that year were all treated instrumentally; 10 were tracheotomized, with a mortality of 25 per cent. The difference in results was obviously due to unremitting teamwork on the part of the bronchoscopists, pediatric staff and special nurses. We have not quite reached Dr. Galloway's record, but the genus epidemicus may have been a trifle in his favor!

It is doubtful if the mortality can be brought much lower. We will always have to contend with the possibilties of complicating pneumonia, sepsis, pneumothorax, unsurmountable exhaustion and the patient who is seen too late.

Dr. Paul Holinger: I was much interested in Dr. Galloway's paper and Dr. Brennemann's discussion. In watching the moving pictures certain things came to mind, although it is hard to add much to what has been said. The child's age is certainly a great factor. Complete obstruction of a lobe or even of an entire lung may be produced by a small mucus plug in the bronchus of a child a year old or less, whereas in a larger child the same size plug would produce few symptoms. Possibly in the older children in whom larger plugs of secretion would block the major bronchi, postural drainage would be of benefit after the secretions were liquefied. In the younger children, however, the secretion is made up of desiccated epithelium which actually replaces the mucosal walls, and it is almost impossible to liquefy it to a point at which it will flow.

This is certainly an interesting subject, and Dr. Galloway should be complimented on his statistics relative to the treatment of these children.

Dr. Thomas C. Galloway: The diagnosis of this condition seems to be difficult, but it often is so because the condition is really not thought of. The diagnosis might also be easier if the laryngologist were called earlier. If there is doubt, one is justified in making a direct laryngoscopic or, if necessary, a bronchoscopic examination. Pediatricians now, I think, have concluded that this can be done without great harm to the patient. By "early," I think Dr. Brennemann and I mean the same thing. These children were tracheotomized from the second to the fifth day. They were closely watched, and when they began to go "bad" intervention was immediate.

It should be emphasized that postural drainage is not a question of an hour or two a day. To be effective it must be continuous or nearly so, especially, as Nelson has shown, for viscid secretions such as one finds in bronchiectasis. In tracheobronchitis probably the secretion is not so viscid and will drain by gravity; surely that is so after irrigation. As Baum showed, crusting usually occurs because of tracheotomy, and I think ulceration is likely to be related to crusting. The postmortem picture is an end result that may not be reached in more favorable cases.

One purpose of the discussion is to emphasize that the picture is not just one of laryngeal and tracheal obstruction but one in which superimposed mechanical and vascular factors make much more complex changes. Although these are not well understood, the indication for earlier and more active intervention seems clear.

Early Diagnosis of Laryngeal Carcinoma. Presented by Dr. Francis L. Lederer.

My presentation this evening is purely informal and therefore unpremeditated. Any virtue it may possess and all its shortcomings arise from the fact that it is given without deliberation. My method of approach may subject me to the scorn of those who will feel with incredulous rage that I fail to dwell long enough on those favorite avenues which they feel are all-important. That is inevitable, but

I hope that they will consider that I offer but my own impression, without bias, guided by the experiences which it has been my good fortune to have—well realizing that I am addressing myself to those whose ideas are rather set and who feel complacently capable of following a pattern. The danger of this, as every exponent of scientific medicine knows, is retardation of progress. When every physician who has an interest in the subject of carcinoma of the larynx, or even in malignant developments in general, realizes that the problem cannot be solved by taking sides medicine will have advanced a long way. Any group unwilling to accept this philosophy constitutes in itself an economic and social cancer. I say these things because I feel so keenly that scientifically and commercially one modality has been pitted against another.

One of the most intriguing studies in medicine is that of the differentiation of disease entities which have a similarity of symptoms. Hoarseness is one of those symptoms; by this is not implied a loss of voice (aphonia) but any interference with the intensity, pitch and quality of the tone.

Hoarseness may be defined as a roughness or a discordance in the quality of a voice influenced either by an alteration of pitch or intensity or both. Numerous anomalies of the larynx are manifest at birth. As man progresses through life there is perhaps no organ which by virtue of its position and function is subject to greater mistreatment or abuse than the larynx. Diseases of the upper and lower respiratory tract, vocal abuse, systemic disease, diseases of the surroundings of the larynx and external irritants are largely responsible for many abnormal manifestations. The causes of such changes are numerous and should be known not alone to the laryngologist but to the general practitioner, who must be made to appreciate the importance of voice changes, and to the layman, who must be made to realize that this symptom is a danger signal of laryngeal dysfunction—they must become "voice and hoarseness conscious."

It must be borne in mind constantly, however, that hoarseness and inspection do not always enable one to establish the diagnosis objectively. Use of the laryngeal mirror must replace the guesswork which is so commonly employed in the diagnosis of hoarseness. It is too often assumed that hoarseness is due merely to an irritant and that a regimen of voice rest and inhalations of tincture of benzoin will clear up the condition. No one would attempt to make a diagnosis of a pulmonary disease without properly auscultating and percussing the chest, or conclude that there was a pathologic condition of the kidney without a urinalysis or a functional test. A complaint referable to the larynx deserves the same approach, viz.: (1) a careful complete recording of the history of the case; (2) indirect examination with the laryngeal mirror; (3) complete general physical examination, particularly of the upper respiratory tract; (4) roentgenologic studies of the sinuses, chest and neck; (5) examination of the blood, both microscopic and serologic; (6) repeated examinations of sputum; (7) direct laryngoscopic bronchoscopic and esophagoscopic inspection, and (8) biopsy.

As a laryngologist progresses through his span of professional activity, he comes to realize more and more that every cause of continued hoarseness must be considered in arriving at a proper diagnosis. While he may tentatively feel that a certain condition is present, it is his desire to diagnose that condition by elimination of those diseases which present analogous laryngeal pictures. He therefore considers the classification of chronic laryngitis:

A. Chronic nonspecific laryngitis
   1. Chronic hypertrophic (diffuse or circumscribed) laryngitis
      (*a*) Simple
      (*b*) Chorditis nodosa
      (*c*) Pachydermia laryngis
      (*d*) Hemorrhagic
      (*e*) Subglottic
      (*f*) Contact ulcer

2. Laryngitis sicca
3. Perichondritis and arthritis

B. Specific laryngitis
1. Tuberculous
2. Syphilitic
3. Scleromatous

C. Neoplastic conditions
1. Benign
2. Malignant

D. Paralysis
1. Functional
2. Organic

In a purely didactic approach to the cause of laryngeal change there is a tendency to enumerate points in favor of each condition with an unjustifiable abandon. It is only by careful elimination of all possibilities that an absolute diagnosis is reached.

Hoarseness which is pronounced, persistent and progressive arouses a suspicion of carcinoma. Indirect and direct laryngoscopic inspection enables the laryngologist to study the lesion for diagnosis and to rationalize treatment. It should be borne in mind that the pathologic changes of the larynx lie in more than meets the eye. An infiltrative lesion is not always established by immobility of the vocal cord.

Experienced laryngologists have a wholesome respect for the site of the lesion as of prognostic significance. A difference in the distribution of lymphatics is responsible for the fact that a carcinoma on the true cord or below it may be present for a long period without manifesting metastatic qualities. Not so with a lesion above this area or on a cord close to the arytenoid cartilage; from either of these sites metastasis is common. The choice of therapy is often dependent on such deductions.

A word about the type of malignancy is not amiss. Let it be understood that for all practical purposes the site of the lesion and what effect the lesion is having on the host (both local and general) are more important than the cellular structure. In my experience the removal of tissue for histologic study is imperative and is not attended by danger of spreading the disease. Removal of tissue by indirect laryngoscopic manipulation whenever possible is advised, and all tissue removed should be sectioned. Numerous bits of tissue are removed at times, and it is necessary to study all, in order that malignancy, which may be absent in some, may not be overlooked. Excision should be thorough, particularly in depth.

Intralaryngeal procedures of any kind, except in the incipient stage, are rejected because they are ineffective and therefore dangerous. For any operable malignant growth, radical surgical removal augmented by electrocoagulation is the only justifiable measure. Laryngofissure preliminary to the removal of the cord, electrodissection or electrocoagulation has proved satisfactory, at least in selected cases of early involvement of the anterior two thirds of the cord. The chances for recovery are absolutely nil when at an early stage thorough removal of the growth cannot be effected. When this procedure can be carried out satisfactorily, the prognosis becomes relatively favorable.

The safest rule for the management of laryngeal carcinoma is surgical treatment if the lesion is near its incipiency and nonsurgical if the lesion is advanced. When on careful examination there appears to be no peripheral involvement and the growth is so situated that it can be, so to speak, punched out with considerable surrounding tissue by special cutting forceps, this may be resorted to under direct laryngoscopic control or by laryngofissure. An ulceration or thickening near or at the edge of a cord may thus be removed. Unfortunately, such cases

in clinical practice are comparatively rare, the laryngologist usually being consulted when the process has advanced too far for this comparatively simple procedure. Complete extirpation of the larynx then is the only procedure affording some chance of recovery. Laryngectomy is now advocated by most experienced laryngologists when the limits of the false cords are not passed, that is, when only the tissues of the true cord proper are involved and when there is no glandular involvement.

Treatment of carcinoma of the larynx is medical or surgical. The management of laryngeal carcinoma by means of irradiation has not been as uniformly successful as that by surgical intervention. My colleagues and I have diligently followed through case after case with the accepted mode of therapy and find ourselves woefully lacking in encouraging statistics. We are awaiting the time when this form of therapy emerges from its stage of empiricism to produce such results that we can have complete confidence in its employment.

It is perhaps unnecessary to state that an important chapter has been added to the annals of surgical advance by the progress which has been made in the treatment of laryngeal carcinoma. No more need the tremendous mortality of the operation be feared, for hopelessness is not justified with a procedure, albeit formidable, which contains the promise of permanent cures. Such results, formerly the experience of but few, are attributable to early diagnosis, made possible by better training of physicians, and to an improved armamentarium, the laboratory facilities for diagnosis and the widespread education of the public to regard pronounced, persistent and progressive hoarseness with suspicion. The operation itself has not been greatly changed in the past decade, but selection of the patient through meticulous examination by the internist and proper preoperative preparation have greatly enhanced its therapeutic result. Newer methods of obtaining rectal anesthesia and the more general use of local analgesia also have materially aided in reducing the danger of the operation, which formerly incurred great mortality.

Rehabilitation of the patient's voice is of utmost importance. Whenever removal of the larynx is suggested for carcinoma, the immediate question is "What about the voice?" because the possible loss of speech is by no means a pleasant thought. The temptation to reply that "dead men do not talk" is suppressed in favor of the humane retort, "Yes, but why place speech foremost, when life itself is endangered? Besides, with a tumor in the larynx, there is no normal or even audible voice." This is said in a sense of fairness to one who has been apprised of a dread disease and cannot think logically in the first shock.

While life is always placed uppermost in the attack on cancer, the laryngologist must nevertheless be in a position to rehabilitate the patient, building up what has been torn down for the conquest of disease. To this end, the patient is informed that it is possible to speak without a larynx through the development of pseudospeech or by the use of an artificial larynx. The latter is a simple device which soon allows the patient to resume his social and business contacts. A number of patients who have undergone laryngectomy begin quite early to exhibit a form of speech, producing consonants with the buccal muscles through bringing the air in the mouth under a certain pressure with the articulatory narrowing. The vowels themselves are heard weakly as appendages to the preceding consonants, the patient using the aftersound of the consonantal murmur. In this type of speech respiration does not enter into play.

LIVING LARYNGECTOMIZED PATIENTS (Slides and Photographs). Presented by DR. JOSEPH C. BECK.

The loss of the voice, however hoarse or however difficult the use of it in speaking, is a most important consideration to the patient when one proposes removal of the larynx. I have found this to be the greatest obstacle and have tried to fight it by having the patient listen to phonograph records and movietones of laryngectomized patients and by having the patient talk to other patients who

have undergone laryngectomy and learned to talk with an artificial larynx. Yet the patient was not "sold on" the operation. I see I am discussing Dr. Lederer's paper rather than my subject, so I had better concentrate now on my own topic.

The material I am presenting is not a group of walking delegates, such as Dr. Lederer showed, but a series of photographs—only four of them—selected over a period of years, from the time I started to do this work in 1905 until 1935. Since that time this major surgical work has been done by my partner, Dr. M. R. Guttman. Those who have seen laryngectomies know that great progress has been made and that there should be a great many more persons living following laryngectomies today than lived following my operations. When I first started this work the two outstanding complications were mediastinitis and pneumonia. Then came a period of conservatism during which laryngologists performed very few laryngectomies but rather laryngotomies, followed by roentgen treatment in about the same dosage which the dermatologist would use to day for a superficial lesion of the skin. Then came McKenty's monumental work. From then on laryngologists began to see benefit from laryngeal resection.

The four pictures shown are examples which I wish to describe specifically. Statistically, I have performed 212 laryngectomies in private practice for cancer of the larynx. Of the patients, 37 remained well without recurrence for more than five years. Those of the 37 that succumbed did so through intercurrent disease, and whenever a postmortem examination was made there was no metastasis demonstrable. Of the 37 with cures, 14 are still living, and it is with a great deal of satisfaction that I say in spite of the unemployment situation 12 of the 14 are earning their own living. I think they are certainly entitled to a pension.

CASE 1.—G. F., a man aged 78, had been given a diagnosis of squamous cell carcinoma of the larynx. He was operated on (laryngofissure) by a general surgeon in one of the largest clinics in the Northwest, in 1920. The surgeon declared the carcinoma inoperable for laryngectomy and advised the man to return home for palliative treatment. Two years later the patient was referred to me by his family physician because of great difficulty in breathing. A hurried tracheotomy was necessary. At the patient's behest and insistence I performed a radical resection of the larynx in December 1921, including tributary glands of the neck and the greater portion of the contiguous part of the esophagus. This patient had a very stormy time following the operation in that he had two attacks of erysipelas which extended to his scalp and downward to the abdomen. I wish to emphasize these attacks of erysipelas in view of the subsequent history of nonrecurrence. This patient has been and still is active as sheriff of his town.

CASE 2.—B. K., a man aged 76, was shown at biopsy to have squamous cell carcinoma of the larynx. Laryngectomy was performed Nov. 1, 1928, in the usual manner, and subsequent examination of the diseased parts of the larynx showed that a fibrotic type of tuberculosis was also present. The patient had never had any pulmonary symptoms of tuberculosis. He made an uneventful recovery. He has had two attacks of osteal tuberculosis with recovery. This patient, like the first one, was never able to use an artificial larynx but depended on the "burping" type of speech.

CASE 3.—H. S., a man aged 61, was given a diagnosis of squamous cell carcinoma. The usual laryngectomy was performed in March 1925, with complete recovery. The unusual feature of this case was the fact that neither the buccoesophageal voice nor the artificial larynx satisfied the patient, and he conceived the idea of improving his speech by poking a hole, with the aid of a red hot ice pick, from the upper part of the tracheal ostium to the back of the tongue. He kept this opening patent by persistent use of a goose quill for several weeks so that the tract never closed. He has learned to close this opening at meal times so that food and drink do not escape into the lower respiratory tract. By means of a finger placed over the tracheal ostium he is able to force sufficient air through and thus produce a more even voice—in fact, the best voice I have ever heard in a laryngectomized patient.

CASE 4.—This case is interesting from three standpoints: First, two varieties of carcinoma were diagnosed; second, the use of the Coutard treatment proved to be apparently futile and, third, use was made of a Levine feeding tube, the patient having refused gastrostomy. The patient was Dr. B., a man aged 64. Seven years prior to his second visit to my office I saw him with a growth on the right ventricular band and advised biopsy. When I saw him seven years later he had been tracheotomized and was unable to swallow even water. There was a great loss of carcinomatous material in the esophagus and larynx. As stated, he refused gastrostomy and, following a morcellement resection of the larynx and esophagus, performed by Dr. Guttman in February 1938, a Levine feeding tube was inserted. The patient made an uneventful recovery and existed on this type of feeding for seven months, when cardiac failure developed suddenly and he died. The first type of carcinoma diagnosed was transitional cell. In this the Coutard type of treatment was used, and correctly so, but it failed. The second mass we removed showed squamous cell carcinoma. Apparently there was no other metastasis about the body, although we were unable to obtain permission for a postmortem examination.

DISCUSSION ON PAPERS OF DRS. LEDERER AND BECK

DR. THEODORE J. WACHOWSKI (by invitation): I came here with no misconception, because I understand Dr. Beck's attitude on the subject, and I am glad that Dr. Lederer has prepared the way for the apologia which I came to give but which I consider unnecessary.

I would remind you of the early days of laryngeal surgery, as reviewed by Douglas Quick, when the nail on the forefinger was the scalpel and the gruesome first attempt at laryngectomy in New York was remarkable because of the fortitude of the patient.

I remind you that radiology is only forty years old and that Coutard therapy is only eighteen years old. Radiation biology is so young that there is still disagreement on some of the important facts, and only recently have investigators learned to measure roentgen rays and radium on a comparable basis.

If one considers the multiplicity of factors in radiation therapy, the necessity for checking the role of each factor individually, the confusion created by the rapid improvement in equipment, and then realizes that even the five year period of cure is not entirely satisfactory, it must be apparent that the evaluation of the statistics is time consuming. This explains the delay of roentgenologists in reaching the definite conclusions that laryngologists demand. I should like to review the situation briefly.

Regaud noted the differential action of gamma rays on the various cell types and Coutard applied this observation to the therapy of malignant lesions of the upper respiratory tract. He found that a beneficial dose had to be close to the limit of tolerance of the tumor bed and that such a dose could not be delivered in less than fifteen days without risk of serious damage. While such a short course was satisfactory for radiosensitive tumors, a longer, less intensive course appeared to be desirable for more differentiated cells, apparently owing to the preservation of the status of the vasculoconnective tissue. Prolongation beyond sixty to ninety days, however, might result in radiovaccination of the tumor.

Large treatment portals reduced the amount of therapy that could be given, because of toxemia and the danger of late radiation damage. Most of the cases in which treatment was successful fell within a certain dosage zone, beyond which cures were rarely effected while the danger of radiation damage was increased.

While the biopsy specimen may give a clew as to the radiosensitivity of a lesion, the location of the lesion has been the best index to probable curability. Roughly, the chance of cure decreases as the tumor spreads, or is primarily located, away from the epithelium of the vocal cords.

Some statistics of cures have been published, the best of which are Coutard's, who reported 20 to 25 per cent five year cures. There is no way of comparing surgical and radiologic statistics, however, because the surgeon chooses only the

patients whose lesions are favorable, while practically all patients are treated, at least to the extent of palliation, by the radiologist. We get patients late at the Research and Educational Hospitals, and our statistics are poor. In order to present any statistics on cures, the patients should be followed to the postmortem table, to be certain that there were no carcinomatous rests.

I think it is entirely justifiable to do what Dr. Brunner tells me was done in Vienna a few years ago. Physicians decided to give Coutard therapy a trial and so treated all their patients with carcinoma of the larynx by that method. That does not mean that they were convinced of radiation's superiority—some were absolutely opposed. But the five years will be completed in a short time, and they can say that irradiation has done this or that, and their statements will have the weight of adequate trial.

DR. MAX CUTLER (read by Dr. Bernheimer): The question of surgical removal and irradiation in the treatment of carcinoma of the larynx can be considered from the point of view of three types of tumors:

1. A large, extensive extrinsic carcinoma which is outside the domain of surgery but which, as a rule, is composed of highly undifferentiated cells and is therefore radiosensitive. This type requires no discussion, as it is admittedly inoperable both technically and biologically.

2. A small carcinoma limited to the true vocal cord, which technically is operable. When microscopic examination of the specimen discloses an adult hornifying squamous cell tumor, and if this tumor has not reached the anterior commissure, surgical removal gives excellent results and is the method of choice.

3. A lesion which affects the true vocal cord and which technically is operable but biologically is inoperable. This group includes about 20 per cent of laryngeal tumors. In spite of their small size and technical operability, they are composed of undifferentiated cells which have extended along the lymphatics anteriorly to the commissure or posteriorly to the arytenoid. Surgical removal of a lesion falling into this group is commonly followed by local recurrence, and the surgeon is surprised to find a local recurrence after removal of a lesion which seemed so favorable and so definitely operable. This group is radiosensitive and responds well to radiation therapy. It would be interesting if the surgical statistics on this group were segregated and an attempt made to determine whether failures do not concern tumors exhibiting this histologic structure. Perhaps this 20 per cent group would yield better results if the lesions belonging to it were treated by radiation. There are no radiation statistics, however, to support this thesis at present.

There is an intermediate type with regard to which a decision between surgical removal and treatment by radiation is difficult. The difficulty may lie in the fact that the lesion is of borderline operability or in the fact that the patient's age or his general condition contraindicate operation. The individual factors in each case must be carefully considered. These factors are histologic and clinical. Histologically, if the tumor cells are highly undifferentiated, they are generally radiosensitive. If the cells are of an adult squamous form, they are generally radioresistant. A more accurate guide to radiosensitivity, however, is the fixation or mobility of the cord. The highly undifferentiated cell type is almost without exception movable even when extensive; the differentiated cell type is nearly always fixed. When a discrepancy exists between the histologic structure and the mobility of the parts, the latter is the more reliable sign.

It is important to recognize that fixation may not be due to neoplasia alone but may be due to inflammation. A moderate amount of radiation invariably results in reestablishment of mobility when fixation is due to inflammation. This response may serve as an accurate and important guide in determining this point and deciding between surgical removal and irradiation in the treatment of borderline conditions.

DR. L. B. BERNHEIMER: My own experience with radiation therapy for laryngeal carcinoma has been much more fortunate than Dr. Lederer's. I have seen at

least 12 five year cures of intrinsic laryngeal carcinoma, and 1 of extrinsic. The patient with the latter result is living and well after five years, without signs of disease.

At the Veterans Administration Facility (Hines, Ill.), we have attained Dr. Lederer's conception of the ideal relationship between the radiologist and the surgeon. Each patient with laryngeal carcinoma is studied both by Dr. Cutler from a radiologic point of view and by myself from a surgical one before a decision is made as to the type of therapy to be employed. We have certain biologic and anatomic principles which guide us in formulating our opinions. The biologic considerations have been indicated both by Dr. Cutler and by Dr. Wachowski, who quoted Coutard. The anatomic considerations briefly are as follows:

1. All primary subglottic malignant growths are treated surgically. Incidentally, the number of primary subglottic lesions seen is much greater than we heretofore believed.

2. Infiltrating lesions resulting in fixation of the involved cords are usually treated surgically. A therapeutic test with 2,000 roentgen units has, however, influenced our decisions. I have seen fixed cords become movable following such a trial, in which event irradiation is continued. When such a cord becomes movable, we feel that the fixation resulted not from actual infiltration by the malignant growth itself but from inflammatory changes due to secondary infection.

3. Extrinsic lesions are usually irradiated. I have seen an extrinsic lesion respond to this treatment to the extent that only the intrinsic primary growth remained. This larynx has then been considered operable and has been subjected to the indicated surgical procedure.

4. Intrinsic noninfiltrating lesions are usually irradiated, especially when their biologic character indicates radiosensitivity.

Sometimes, however, factors which are beyond our control determine the types of therapy. Such a factor is an intercurrent infection of the upper respiratory tract during a course of radiation therapy. Such an infection may cause marked local reactions, necessitating the substitution of surgical for radiologic therapy. We have also encountered patients who refused surgical operation. These patients have preferred to chance radiation therapy rather than submit to laryngeal surgical operation even after it was explained that in their particular instance surgical treatment offered a better chance for cure.

DR. FRANCIS L. LEDERER: Since I talked on early carcinoma, I naturally left out many procedures, such as lateral pharyngotomy and the like, which are applicable in extrinsic lesions.

I wish to call attention to a case published by Dr. John E. McKenty (Laryngeal Cancer: Early Diagnosis and Treatment, ARCH. OTOLARYNG. **9**:237 [March] 1929), a case that from 1922 to 1927 went through the gamut of every type of diagnosis before the growth assumed a most characteristic appearance. Laryngologists could all study that case very carefully and profit thereby, because it typifies what often happens. I did not mention the use of roentgen rays, applied preoperatively and postoperatively. We employ these rays postoperatively to get any stray cells that may be present. I would warn you about leaving a permanent rubber feeding tube in situ for a long time. I remember a case of laryngeal carcinoma cured with radium. The destruction of tissue which followed irradiation was so great that the man had to be fed by nasal catheter. It is interesting to note that my associates and I found positive evidence of malignant growth despite the tremendous irradiation.

The term "radiosensitivity" must be qualified. Today I saw a patient with laryngeal involvement who responded excellently to irradiation only to suffer from recurrence. One should bear in mind that radiosensitive tumors may recur radioresistant and that they offer a tremendous problem so far as future treatment is concerned. Subglottic carcinoma is of great interest and occurs more frequently than is realized. In the French literature there is recorded a per-

centage strikingly high; I believe in 25 per cent of the cases carcinoma of the larynx was subglottic. In addition, carcinoma occurring in the laryngeal ventricle should have attention because the diagnosis is sometimes not established until there has been direct laryngoscopic inspection.

I was pleased to hear Dr. Cutler's comment, yet I believe it was irrelevant so far as my remarks were concerned. Many points have been advanced this evening, and, I believe had Dr. Cutler been here, he would have taken an entirely different approach. It is absolutely worth while to have a laryngologist working with the specialist in irradiation. It would be interesting to have patients with five year cures presented in person, with laryngeal pictures and biopsy evidence. However, if all the statistics are as bad as ours, especially with reference to the follow-up—I wonder if some of the 18 persons who are described as cured by Dr. Bernheimer are not some that visited our clinic or some one else's with recurrences. If the patients cited are cured even over a five year period, laryngologists must doff their hats to him and consider that something worth while is being done for laryngeal carcinoma and that the future promises much for radium and roentgen rays. However, so long as I can promise more certain results with surgical intervention I shall continue to advise surgical treatment for laryngeal cancer until the other modalities can supply better statistics.

THOMAS W. LEWIS, M.D., *President*, Presiding

Regular Monthly Meeting, Nov. 7, 1938

WALTER H. THEOBALD, M.D., *Secretary*

EXPERIENCES WITH A VITAL STAIN ON OTOLARYNGEAL TISSUES. Presented by DR. ELLISON L. ROSS.†

The material presented in this report is included in an article which appears in this issue of the ARCHIVES, page 428, by Drs. Ellison L. Ross† and I. W. Hamilton, entitled "Penetration of the Inner Ear by Mercurochrome."

DISCUSSION

DR. J. R. LINDSAY: I listened with interest to Dr. Ross's paper, which gives further results of his investigations on the ear, and I think he is to be congratulated on following up this line of work. The evidence presented here that mercurochrome will pass through the epithelium and mucosa of the middle ear or the mucosa of the nose and sinuses and even through the bony walls of the frontal sinus and the overlying dura in such a short space of time is at least surprising. The question which obviously arises and which Dr. Ross has been trying to answer is: How does the drug pass through? Is this a matter of simple diffusion? Or does the extension proceed to the deeper parts by way of lymphatics or capillaries?

Dr. Ross has done other experiments, placing various substances, both crystalline and colloidal, in the middle ear and studying the effect on the function of the labyrinth.

My colleagues and I have also used some of these substances and have been unable to explain why substances such as strong salt solution when placed in the middle ear produce deafness and other substances which we have used do not cause deafness—for example, dextrose solution or plaster of paris when sufficiently diluted so that it does not become solid. The whole matter of chemical changes which occur in the internal ear, and also of physical changes, has rather successfully resisted investigation.

† Dr. Ross died on Dec. 21, 1939.

It is known that some crystalline substances will not pass through living membranes—for example, phenolsulfonphthalein injected into the sac of a hydrocele does not pass through the walls. In the case of mercurochrome it seems that the action is simply diffusion, in which the substance is not confined to lymph or blood vessels but spreads through all tissue. The rapid diffuse spread of the drug throughout the mucosa and even the bony walls might again raise the question of the therapeutic value of this drug as an antiseptic. However, the fact that the drug diffuses into the tissues cannot be taken as evidence that it could be present there in sufficient concentration to have therapeutic value or that it would not have a detrimental effect, for example, on the action of the cilia. It is true that chemotherapy has received a boost in recent years in the discovery of the action of sulfanilamide, but from the evidence to date the beneficial effects obtained by chemotherapy seem to be limited to a few drugs, most of which apparently have a specific effect on certain types of organisms.

Dr. George T. Jordan: I should like to ask the method of instillation. Was the mercurochrome put through the tympanic membrane with a hypodermic needle or merely dropped onto the drum?

Dr. Samuel J. Pearlman: The penetration of the mercurochrome through the mucosa and bone into the dura is to me an astonishing thing. There is one matter not quite clear to me. If I understand it correctly, the mercurochrome was placed in the nose and after a certain time the animal was perfused with a mild acid solution for several hours to several days. After awhile the animal had in its vessels and tissues only the dilute acid solution. The question is: Is it possible that some of the drug was dissolved and carried in through bone and to the dura by the blood vessels and deposited where it was found? I should like to ask whether control experiments were made. Were the tissues sectioned after staining and without the perfusion? It is easy to see mercurochrome in the tissues, and how can one tell that the remarkable depth of penetration was not due to the methods used?

Dr. Ellison L. Ross: Dr. Jordan has asked how the compound was applied. A blunt needle was put through the ear drum and the mercurochrome injected into the middle ear.

Dr. Pearlman has asked whether the perfusion might not have carried the stain into the tissues. The fact that the depth of the penetration of the tissues was the same in cases of varying thoroughness of perfusion argues against the carrying of the stain by the injected solution. Mercurochrome seemed to have passed into the tissues much in the manner of diffusion. The part played by the circulation is not yet determined. It was hoped that, knowing the mode of travel of mercurochrome in tissues, one might be guided in the use of therapeutic agents intended for deep tissues.

Inflammatory Diseases of Meninges and Brain of Pharyngeal Origin. Presented by Dr. Hans Brunner (by invitation).

Three cases are described, in one of which the base of the skull was examined thoroughly macroscopically and microscopically. From these studies the following conclusions have been drawn:

1. In the course of sepsis having its origin in the pharyngeal region the meninges and brain may be affected (*a*) by means of a phlegmon of the parapharyngeal space or (*b*) by means of general sepsis.

2. Infection of the parapharyngeal space may occur with a critical reduction of temperature and increasing leukocytosis, but without alarming general or local symptoms.

3. Neuralgia of the trigeminal nerve can be absent in cases of phlegmon of the parapharyngeal space.

4. The locking of the eustachian tube in phlegmon of the parapharyngeal space occurs as a result of infection passing along the connective tissue and along the

blood vessels, which under normal circumstances perforate the cartilage of the tube, passing from the parapharyngeal space into the mucous membrane.

5. At operation on deep abscesses of the neck one should not incise a normal sheath of the blood vessels if there is purulent necrosis in the surrounding fascia of the neck.

6. When infection of the parapharyngeal space reaches the contents of the skull, first the meninges become infected, and then intradural suppuration and thrombophlebitis of the cavernous sinus and of the carotid plexus develop. All these diseases of the meninges are, as a rule, accompanied by typical, familiar symptoms, but sometimes these symptoms may be missed. Contrary to those infections produced by way of a phlegmon of the parapharyngeal space, the general sepsis first affects the brain itself, while infection of the meninges either does not occur or is of secondary importance.

7. The infection of the brain in general sepsis may lead to various pathologic entities. In the cases reported here edema of the brain was produced.

8. This septic edema of the brain may appear clinically as a disease localized in the posterior fossa.

9. Septic edema of the brain may disappear with recession of the sepsis originating in the pharyngeal tissues.

DISCUSSION

DR. SAMUEL J. PEARLMAN: To say that I enjoyed this paper would be putting it mildly. Apart from the exceedingly interesting subject, the vivacious manner and enthusiasm with which Professor Brunner presented this study gave me a great deal of pleasure.

Otolaryngologists have known for a long time that the parapharyngeal abscess has a tendency to travel downward into the mediastinum. They know that in such a case it is necessary to drain the neck and use the tamponade of Marshik to prevent descent into the mediastinum. However, I am certain that from time to time the literature will present cases of parapharyngeal abscess in which the reverse situation will be called to attention just as forcibly; that is, the process may travel upward to the base of the skull and kill by meningitis. It should surprise no one if a patient who has tonsillitis, then peritonsillar abscess and finally parapharyngeal abscess should die of meningitis. I should like to trace again some of the pathways which have already been mentioned, as much for my own benefit as for any one else's. I ask, how does the infection travel, and the answer is that there are a number of pathways which should be obvious to any one who considers the anatomy involved. Pus deep in the neck goes as high as the base of the skull. It is only one step to go through the basilar foramina, particularly the foramen ovale, foramen spinosum and foramen lacerum medium. The infection may attack the bone and produce meningitis by direct extension. That would be a long-standing process and is a theoretic consideration only. By the time the bone at the base of the skull is involved enough to permit extension to the meninges, the patient will probably be dead or recovered from his parapharyngeal infection.

There is another route by which the brain may be involved, and here are the pathways as I know them: It is possible for the retrotonsillar veins to become involved; from here the infection may extend to the jugular vein and the jugular bulb and thence to the cavernous sinus by way of the inferior petrosal sinus. This development is not common but does occur, and death takes place by meningitis from involvement of the cavernous sinus. Or, from the retrotonsillar veins there is a spread to the pterygoid plexus; once this is involved the cranial cavity is attacked by way of veins which go through the foramina spinosum, lacerum medium and ovale to the cavernous sinus; or the spread may occur in the form of thrombophlebitis from the tonsils to the pterygoid plexus, through a deep communicating vein, then to the anterior facial vein, to the angular vein and finally through the orbit by way of the ophthalmic veins to the cavernous sinus.

There is another way—directly by way of the walls of the carotid artery. Ira Frank reported infection in the walls of the carotid artery up to and into the brain. Still another route is by way of the plexus of veins which surround the carotid artery. These become involved from the pus which bathes the carotid artery or from a thrombosed internal jugular vein which communicates with the carotid venous plexus. The cavernous sinus is then attacked by extension through veins leaving the foramen lacerum medium.

One should not be surprised that in this patient the usual signs of cavernous sinus thrombosis were absent. Eagleton a long time ago called attention to the fact that the cavernous sinus may be involved from in front, medially and from behind. The fixation of the bulb and the orbital cellulitis which one associates with classic cavernous sinus thrombosis is seen chiefly in cases in which the sinus is involved from in front by way of the orbit. So that if the cavernous sinus is involved from behind the orbit one cannot expect to observe fixation and protrusion of the bulb until very late.

There is another point which should be brought out in these pictures of infected carotid arteries. Not only meningitis can occur, but an abscess of the brain. The infected clot in the carotid artery may be loosened and then it may lodge in the brain to produce an abscess.

I should like to thank Dr. Brunner for his fine presentation. The time is coming, and this is one of the occasions, when physicians will become increasingly aware that parapharyngeal infection travels upward as well as downward and can kill by invading the brain just as it does by going downward to produce mediastinitis.

DR. SHERMAN SHAPIRO: I have reason especially to express my admiration for the masterly manner in which this first case report was presented.

Some of the members may recall that several years ago I presented a study of parapharyngeal infection following tonsillectomy. At that time I combed the literature very thoroughly but never found record of anything like this; tracing the pathways of infection. I do not know why some infections travel upward and not downward. I do not know whether any one can answer that. Among the cases I reported there were 3 fatalities, 1 from meningitis. No permission for postmortem examination was obtained. It is evident that the percentage of patients who die from complications involving the brain is considerably smaller, so that the more common route is apparently downward.

I was interested especially in Dr. Brunner's point in regard to edema of the brain. Within the last two years I have seen 2 patients, referred by the neurosurgical department, with symptoms of disease of the pontile posterior fossa. One had hemiplegia, a child aged about 12. Both patients were seen long after the initial focus of infection had developed—in one case it was mild otitis, which passed off, and in the other, an infection of the upper part of the respiratory tract. The disease of the initial focus had already passed, and my associates and I were dealing strictly with the intracranial phase of the situation. I never thought of edema of the brain; I thought there was an area of encephalitis, as mentioned by Dr. Brunner. The second patient was recovering, and the hemiplegia was subsiding. The other patient was sent home in poor condition, and I do not know the outcome. It is certainly worth while to think of edema.

Another condition to consider is arachnoiditis. In any involvement of the posterior fossa one must think of circumscribed and other collections of cerebrospinal fluid, which may contribute to tumor of the brain.

DR. NOAH SCHOOLMAN: I rise to take part in this dicussion with the thought that fifteen years ago I had the privilege of listening to the lectures of Professor Brunner in Vienna, but had no opportunity to talk back, and two years ago my son had the same privilege. I take the liberty now to emphasize the close relation of the temporal bone to the pharynx. Aside from the fact that the middle ear communicates with the pharynx by way of the eustachian tube, the apex of

the petrosa as it enters the angle between the basilar process of the occipital bone and the body of the sphenoid invades the pharynx to a point anterior to the plane of the pharyngeal tubercle and thus becomes a part of the bony framework of the pharynx. Under this circumstance it is conceivable that a petrosal abscess may point into the pharynx and may be drained through that route. Some of the pharyngeal abscesses of obscure nature reported in the older literature as of otitic origin may have been of this source. It is therefore of interest to reflect that the presence of an abscess in the pharynx of uncertain origin in a patient exhibiting symptoms of intracranial disease may be a sinking abscess of intracranial origin or a pharyngeal abscess which has extended upward. The difference would be important in the treatment—if the former, one would drain through the pharynx and hope for the best; if the latter, one would be led to intervene intracranially and expect the worst.

DR. HANS BRUNNER: I thank you very much for your interest in my paper, and particularly Dr. Pearlman for his interesting views about the ascending infection.

An infection of the meninges from a parapharyngeal abscess is dangerous, because even when one finds the abscess and can open it, it is practically impossible to eliminate all pathways to the skull and the contents of the skull, since the ascending infection travels as a rule by more than one pathway. That is the first point to be emphasized. The second is that in these microscopically proved cases, the conditions must be carefully examined, for when one finds pus in the carotid sheath or elsewhere, it is difficult to determine whether the infection is traveling from below upward or from above downward. Therefore the interpretation of the microscopic picture is difficult. The third point is that the patient who dies of what is thought to be a parapharyngeal abscess with descending infection but who has not corresponding evidences in the mediastinum at autopsy should be further examined especially with regard to the brain, for it is surprising how often the cavernous sinus will be found to be involved without having produced clinical symptoms of such a process.

Dr. Shapiro spoke of cases in which otolaryngeal infection was associated with general increase of intracranial pressure and symptoms of local disturbance in the posterior fossa, often thought to be encephalitis but which in reality is often nothing more than edema of the brain. It is well to remember that generalized brain hypertension may appear to be a disease localized in the posterior fossa, as my cases have shown.

INTRINSIC CARCINOMA OF THE LARYNX: A NEW INSTRUMENT MODELED AFTER THE JACKSON BRONCHOSCOPE FOR THE INTRALARYNGEAL APPLICATION OF RADON. Presented by DR. FRANK E. SIMPSON.

This demonstration of a new instrument for intralaryngeal irradiation was given to create interest among laryngologists who have the opportunity of seeing cancer of the larynx more frequently than other physicians, and also to induce others to try this method. The instrument was shown, the technic of administration described, and a motion picture exhibited demonstrating the treatment of the patient whose case is reported.

This woman, aged 80 years, became hoarse in October 1937. In March 1938, when she could hardly speak above a whisper, a diagnosis of squamous cell carcinoma, grade 2, was made. The lesion was on the right vocal cord, in the anterior third. Both cords were freely movable. Radium treatment was begun on April 7; between that date and May 5, 5,000 millicurie hours was given to the cutaneous surface over the larynx with the radium bomb at 4 cm. distance. On May 1 the patient's singing and speaking voice was restored and has remained unimpaired. The vocal cords appeared normal and have remained so up to this time. A phonographic record of the voice made on October 6 revealed a normal voice free from huskiness.

The advantages of the intralaryngeal method are:

1. It is nonoperative, a desirable feature in dealing with elderly and feeble patients.

2. No pain is experienced.

3. The patient's breathing is free and unobstructed during treatment.

4. The radon is held at a fixed distance—5 mm.—from the surface of the tumor, so that dosage is accurate.

5. Danger of injury to the larynx is minimized.

6. If irradiation fails, operation can still be resorted to if advisable.

DISCUSSION

DR. JOSEPH C. BECK: The old saying—the proof of the pudding is the eating thereof—is apropos here. These records show the patient after treatment. What should have been shown was a record of her voice before treatment was begun so that one could actually know. One has the word of the operator that the patient was hoarse, perhaps not as hoarse as I am now, after about ten days of laryngitis.

The lesion was of grade 2. One has heard that the grading in treatment with roentgen rays and radium does not amount to so much after all. There have even been cases of carcinoma that responded to treatment with rays of high voltage, both roentgen rays and radium, but the holding of the radon or any instrument for the length of time mentioned would be rather difficult in some cases. Yet I see where this work surely can be enlarged on—that one could anesthetize the larynx by blocking the superior laryngeal nerve, and have no great difficulty in holding the instrument. Having suction to get rid of any secretions that might accumulate would be of additional help.

Otolaryngologists are still being told definitely that a grade 1 or grade 2 lesion of the larynx, that is, an intrinsic laryngeal lesion, is a matter for operation only—because one is dealing with cancer. Yet in time only will this splendid work that has been shown be verified; time will show just how good the treatment is. But that a laryngologist, as Dr. Hagens is, is working along these lines, gives me a great deal of confidence that the work may become of distinct value.

When I read the program I was prepared for a different discussion. I thought Dr. Simpson was going to tell the members how he would get rid of them as laryngologists by the employment of a direct laryngoscope. This has been a wonderful discussion, and the members should all be very happy that they have heard it and seen the demonstration.

DR. THOMAS C. GALLOWAY: Otolaryngologists have seen carcinoma treated with relatively small doses, with excellent immediate results, indicating a cure by radium, and I think that later disappointments in such cases have made some of them a little skeptical about certain reports. All, I think, are open minded about the results they may get in carcinoma of the larynx with radium, but the proof is in the cures that have lasted five years or longer, and I think until physicians get such results they must suspend judgment.

It seems to me that by this method a not very adequate dosage is applied to the tumor, especially when one considers that the radium might not be absolutely accurately placed in relation to the lesion. It is known, of course, that irradiation short of cure is likely to make a cancer resistant to later irradiation and may carry the patient past the time when other treatment is possible, but this method of treatment is certainly a valuable contribution which can be developed further. Dr. Hagens could perhaps devise some means whereby the tube could be curved or angulated so that it might be tolerated for a longer time; in dealing with some benign lesions, such as multiple papilloma of the larynx, I think it might be extremely valuable, and I hope it will be.

DR. FRANCIS L. LEDERER: I do not think one can pass the subject without thinking of a former member of this society, Dr. Otto Freer. All recall Dr. Freer's

enthusiasm for the direct application of radium to the larynx. I believe Dr. Beck will remember the case in which Dr. Freer made freehand drawings of the larynx to show the tumor disappearing under direct irradiation, only to recur after a length of time, and then, with no evidence of a local lesion but with metastatic extension. Therefore I do not believe Dr. Simpson intends that any one should think, nor should any one accept the thought, that he is presenting this woman as cured. He is merely bringing before the members a new instrument which is certain to be advantageous. Of course, in treating an 81 year old patient such an instrument is very advantageous, because one would not care to entertain operative intervention in such a patient, and if it offers the advantages pointed out, I believe it is a worth while instrument to follow through.

Relative to its being held in place for ten minutes, I recall the times I have tried to hold even a laryngoscope in place to let five or six students look through it, and I know how I failed to hold it accurately for even the length of time the first student looked through it. I am wondering if Dr. Galloway is not right in that it might be difficult to hold the instrument exactly in place with respect to the lesion, especially if there is respiratory motion of the vocal cords, which might alter the direction of the radiation so that one would have to change the position of the radon from time to time.

With respect to the dosage, while it is not high, it has the advantage of being direct and not through tissue. I assume the dose might be thought of as sufficient inasmuch as there is no filtration other than that provided by Dr. Simpson.

I appreciate the opportunity of seeing again that the radiologist has the cooperation of the laryngologist in the treatment of his patients.

Dr. Paul Holinger: This method of applying radon is extremely interesting. I think Dr. Simpson has made possible a real advance in the treatment of lesions of this type, and the method should have further application in diseases of the bronchi and esophagus. The ease of application of radon in this manner suggests the possibility that one might depend on the use of radon instead of surgical intervention in certain cases. The choice in this particular case is extremely fortunate, since the age of the patient rather contraindicates a surgical operation, although statistics still favor the surgical removal of intrinsic lesions.

Dr. Walter Theobald: I should like to ask a question which might be in the form of a suggestion. In screening, is it possible that the radiation might be too great for the unaffected side, and in such a case could one screen half of the tube so that the entire dose would be directed to the affected side?

Dr. Frank E. Simpson: So many points have been brought up that it will be difficult to discuss them all.

My associates and I have thought about additional screening for the normal side of the larynx. This is not feasible, partly because of the necessity of air space for breathing. Moreover, 1 mm. of gold screens off 99.9 per cent of the hard beta rays, and even an additional millimeter would screen off but a very small percentage of those that are left.

So far as the dose is concerned—it seems small and could doubtless be increased. We were extremely cautious so as not to hurt the larynx and really gave less than the toleration dose, knowing that if the dose proved insufficient we could add to it. Our dose proved to be sufficient, however, as the tumor disappeared.

I was associated with Dr. O. T. Freer a good many years ago and helped him treat his patients. Recently, in looking up our records I found we treated 19 persons. The subsequent history of these persons I do not know, because they were Dr. Freer's patients, and his death prevented a proper follow-up. I do know, however, that while patients did well temporarily, some had necrosis of the cartilage and their lesions terminated fatally. We made the mistake in those days of trying to give too much radiation at one time, sometimes as high as 800 millicuries in the larynx for thirty minutes—that is, 400 millicurie hours. Moreover, the instrument used at that time did not hold the radon at the proper distance from

the tumor. I do not believe that a ten minute exposure tends to favor metastasis. We use a large quantity of radon for a short period of time so that there is a minimum of traumatism.

We did not use the suction apparatus because of the possibility of disturbing the carcinoma and because the morphine and atropine administered prior to treatment seemed to check the secretions sufficiently.

We made the gold capsule containing the radon 4.5 cm. long so as to allow for a certain play up or down. If you take three points, one opposite the middle of the first tube, one between the two tubes and one opposite the middle of the second tube and have these three points equidistant from the gold capsule and in a line 5 mm. from and parallel with the gold capsule, the radiation received at these three points is practically uniform. The outside surface of the instrument is marked in centimeters so that the distance from the upper teeth to the larynx can be noted. The operator inserts the instrument until the distal end is at the tumor and then pushes the instrument down 2 cm. more, which brings the center of the gold capsule opposite the tumor. The vocal cords fix the instrument at the lower end, and the operator has little difficulty in holding it immobile for the short time required for treatment.

Directory of Otolaryngologic Societies *

FOREIGN

Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Dr. Louis Ledoux, Brussels, Belgium.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg 143, Groningen, Netherlands.
Place: Brussels, Belgium. Time: September 1939.

Hungarian Otolaryngological Society

President: Dr. J. Treer, Bocskay-u. 9, Szeged.
Secretary: Dr. G. Kelemen, Reáltanoda-u. 9, Budapest.

Sociedad Rioplatense de Oto-Rhino-Laringología (Argentine Section)

President: Dr. Raul Becco, B. Mitre 1690, Buenos Aires.
Secretary: Dr. Juan Manuel Tato, Santa Fé 1171, Buenos Aires.

Société Française d'Oto-Rhino-Laryngologie

Secretary: Dr. Henri Flurin, 19 Avenue Mac-Mahon, Paris, 17°.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Leroy A. Schall, 270 Commonwealth Ave., Boston.
Place: St. Louis. Time: May 15-19, 1939.

American Academy of Ophthalmology and Otolaryngology

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha
Place: Chicago. Time: Oct. 8-13, 1939.

American Bronchoscopic Society

President: Dr. John D. Kernan Jr., 120 E. 75th St., New York.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.
Place: Westchester Country Club, Rye, N. Y. Time: May 27, 1939.

American Laryngological Association

President: Dr. George B. Wood, 504 Physicians' Bldg., Philadelphia.
Secretary: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Place: Westchester Country Club, Rye, N. Y. Time: May 24-26, 1939.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Harold I. Lillie, Mayo Clinic, Rochester, Minn.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Drake Hotel, Chicago. Time: May 9-11, 1939.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

SECTIONS:

Eastern.—Chairman: Dr. Frank E. Kittredge, Masonic Temple, Nashua, N. H.
Southern.—Chairman: Dr. Francis E. LeJeune, Maison Blanche Bldg., New Orleans.
Middle.—Chairman: Dr. T. R. Gittins, Davidson Bldg., Sioux City, Iowa.
Western.—Chairman: Dr. Frederic G. Sprowl, Medical Arts Bldg., Spokane, Wash.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Isidore Friesner, 36 E. 73d St., New York.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.
Place: Westchester Country Club, Rye, N. Y.: Time: May 22-23, 1939.